Updating vs. Exposure to Modify Responses to Traumatic Stimuli
An Experimental Study

Pile, Victoria

Awarding institution:
King's College London

Title: The impact of smooth muscle ageing upon actin cytoskeleton organisation, adhesion and motility

Author: Victoria Plle

Volume I

MAIN RESEARCH PROJECT & SERVICE EVALUATION PROJECT

Victoria Pile

Thesis submitted in partial fulfillment of the degree of Doctorate in Clinical Psychology

Institute of Psychiatry, King's College London

May 2013

Acknowledgments

Firstly, I would like to thank Dr. Jennifer Wild for her supervision of my main research project, her guidance over the course of the project has been invaluable and I have greatly appreciated her enthusiasm and support throughout. I would also like to extend my gratitude to Dr. Patrick Smith for his greatly valued clinical and research expertise. In addition, thank you to Thorsten Barnhofer for his help with the psychophysiology, Andrew Brand for his technical support and Dr. Daniel Stahl for his advice with the statistics. I am also very grateful to all my participants who gave up their time to take part in the research project.

I would also like to acknowledge my clinical placement supervisors: Dr. Rhiannon Owen, Dr. Laura Lunt, Dr. Maxine Sinclair, Dr. Anna Redfern, Dr. Sean Perrin, Dr. Susanna Cole, Dr. Hannah Mollitor, Dr. Alice Kerr, Dr. Nicola Reynolds and Dr. Eric Morris. Their knowledge and skilled supervision means that I have learnt a great deal from each varied experience.

I would like to thank my course-mates for all their support, friendship and wise words. I am also incredibly grateful to Nick and my family for their ongoing encouragement and faith in me over the last three years.

Contents

Main Research Project

Updating vs. Exposure to Modify Responses to Traumatic Stimuli: An Experimental Study

Supervisor: Dr. Jennifer Wild

Second Supervisor: Dr. Patrick Smith

Abstract

Re-experiencing symptoms are a hallmark feature of Post-Traumatic Stress Disorder (PTSD). They are postulated to result from the way in which a trauma memory is encoded, organised and retrieved (e.g. Ehlers, Hackmann, & Michael, 2004). Research has illustrated the effectiveness of exposure therapy (Foa et al., 1991) and Trauma-Focused Cognitive Behavioural Therapy (NICE, 2005) in the treatment of chronic PTSD, yet evidence for early intervention has been mixed. Reducing the fear response is a central aim in therapies for PTSD. Studies have shown that the fear response can be conditioned in humans in the laboratory, that it can return following extinction and that it can be prevented from returning (Schiller et al., 2008, 2010).

This analogue study investigated methods to reduce the conditioned fear response, intrusion frequency and PTSD symptoms after viewing trauma films. The research used an experimental design that combined conditioning and trauma film paradigms. All participants underwent the same fear conditioning paradigm where trauma film stimuli (unconditioned stimuli) were paired with neutral stimuli (conditioned stimuli). Participants were randomly allocated to one of three US devaluation groups: 'update', 'exposure' and 'neutral'. Exposure and updating techniques are frequently used as components of psychological therapy for PTSD but their relative effectiveness is unclear. This study aimed to compare the effects of updating the meaning of the trauma films (update group), further exposure to the trauma films (exposure group) and viewing non-traumatic films of related content (neutral group) on the reduction of the conditioned fear response and analogue PTSD symptoms. This study also investigated whether individual differences in fear conditioning are associated with the development of PTSD symptomatology.

Overall, the findings suggest that adding a cognitive update to a US devaluation process significantly reduces subjective distress ratings to fear conditioned stimuli as well as intrusion frequency and PTSD symptoms. In this study, having a larger conditioned acquisition response predicted higher intrusion frequency and distress and more PTSD symptoms. However, in contrast to the hypotheses, adding a cognitive update to US devaluation increased skin conductance response to the conditioned stimulus compared to further exposure to the films. Theoretical and clinical implications are discussed as well as limitations and avenues for future research.

Contents

Figures

Tables

1. Introduction

1.1. Post-traumatic stress disorder

1.1.1 Diagnostic criteria, clinical characteristics, course and prevalence

Post-traumatic stress disorder (PTSD) is a distressing and debilitating anxiety disorder. PTSD is defined by the development of symptoms in three distinct clusters following trauma. To receive a diagnosis, these symptoms must persist for longer than one month and cause significant impairment in social, occupational, or other important areas of functioning (American Psychiatric Association [APA], 1994). The Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) specifies that the person has been exposed to a significantly distressing event and that the person's response has been one of intense fear, helplessness or horror (APA, 1994). The three symptom clusters described by DSM-IV (APA, 1994) are re-experiencing, avoidance/numbing and hyper-arousal. Re-experiencing symptoms are where the individual involuntarily experiences distressing aspects of the traumatic event again, usually in the form of intrusive repetitive images or thoughts, flashbacks or nightmares. These symptoms are the hallmark feature of PTSD and cause intense distress and physiological reactions in the person (National Institute of Clinical Excellence [NICE], 2005). The avoidance/numbing cluster currently describes behavioural and cognitive avoidance of trauma-related cues (e.g. avoiding talking about the trauma) and/or emotional numbing or detachment from others. This cluster will be separated into two (avoidance and persistent negative alterations in cognitions and mood) when DSM-V is published in May 2013 (Kupfer, Kuhl, & Regier, 2013). The hyper-arousal cluster includes hyper-vigilance to threat, poor concentration, sleep difficulties and irritability.

PTSD is common, with lifetime prevalence rates of 7.8% using DSM-III-R criteria (Kessler et al., 1995) and point prevalence rates of 1.5-3% for adults (NICE, 2005). However, estimates vary depending on what criteria were used, for example in assessment (e.g. definition of traumatic event, measures) and sampling (e.g. age, location). Clinically meaningful levels of functional impairment can also result from sub-threshold PTSD symptomatology, with one-month prevalence rates of partial PTSD being 3.4% for women and 0.3% for men (Stein et al., 1997).

PTSD has serious consequences for the individual and society; symptoms of PTSD impact on an individual's social, educational and occupational functioning and increase the person's risk of developing comorbid physical and mental health difficulties (e.g. Brunello et al., 2001; Hidalgo & Davidson, 2000; Magruder et al., 2004). PTSD is associated with significant economic burden to the families, the National Health Service and society more generally (NICE, 2005; Greenberg et al., 1999). Studies in the United States found that PTSD, via work impairment and increased health service utilisation, was associated with higher costs than any other anxiety disorder (Greenberg et al., 1999).

PTSD has high rates of comorbidity, with lifetime comorbidity rates ranging from between 62-92% (Breslau et al., 1991; Davidson et al., 1991; Kessler et al., 1995; Shore, Vollmer, & Tatum, 1989). Common comorbid disorders include substance misuse, depression and other anxiety disorders such as panic attacks (NICE, 2005). In some groups, comorbidity has been found to be the rule rather than the exception, for example 98.8% of theatre veterans with PTSD had a comorbid disorder compared to 40.6% of those without PTSD (Kulka et al., 1990).

PTSD is not a definite consequence of trauma nor do PTSD symptoms automatically result in long-term difficulties. It is estimated that the risk of developing PTSD following a traumatic event is 8.1% for men and 20.4% for women (Kessler et al., 1995) and that approximately 25-30% of people involved in road traffic accidents develop PTSD (NICE, 2005). PTSD is more likely to occur following combat and acts of intentional violence, particularly sexual assaults, rather than accidents or disasters. In addition, PTSD symptoms following trauma may be a sign of normal adaptation (e.g. Steil & Ehlers, 2000) with most people developing symptoms but the majority recovering without treatment. There is a sharp decline in PTSD rates in the first year following trauma (Breslau et al., 1991; Kessler et al., 1995) and the severity of PTSD symptoms from around 1-4 weeks appears to be a good indicator of treatment necessity (e.g. Harvey & Bryant, 1998; O'Donnell et al., 2007; Shalev et al., 1997). Approximately one third of people who develop PTSD will remain symptomatic for three years or more (Kessler et al., 1995).

1.1.2 Intrusive memories and PTSD

Involuntary highly emotive and intensely distressing memories about the trauma are the hallmark feature of PTSD and the most consistently reported symptom following a trauma (Durham, McCammon, & Allison, 1985; Genest et al., 1990). Holmes & Bourne (2008) define intrusive memories as "involuntary recollections relating to events that appear, apparently

spontaneously, in consciousness". They can be exceptionally vivid, lack time perspective and context and experienced as if the trauma is reoccurring in the present (e.g. Ehlers, Hackmann, & Michael, 2004). Intrusions play a principal role in the prominent cognitive theories of PTSD (e.g. Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000) and are suggested to play a fundamental role in the formation of other symptoms and maintenance of PTSD (Ehlers & Steil, 1995; Michael et al., 2005). For example, Ehlers & Clark (2000) link the distressing experience of current threat resulting from intrusions with hyper-arousal symptoms and the use of maladaptive strategies (e.g. avoidance). Evidence suggests that with therapy the frequency, vividness and distress caused by intrusions decreases (Hackmann et al., 2004).

Intrusions following trauma have been shown to be qualitatively different from autobiographical memories, including being more vivid recollections and having a greater impact on mood (e.g. Berntsen, 2001). Intrusions can be triggered by a wide range of internal and external cues which may relate to moments signalling the traumatic event is about to happen, become more threatening (Hackmann et al., 2004) or the part with the highest emotional impact (Holmes, Grey, & Young, 2005). Ehlers, Hackmann, & Michael (2004) suggest that intrusions represent the re-experiencing of warning signals for future threat but evidence for this has been mixed.

Most people will develop intrusions following trauma and high levels of intrusions predict PTSD 12 months later (O'Donnell et al., 2007). Distress resulting from the intrusions, their lack of context and the sense that they are occurring in the present have also been shown to be good predictors of PTSD (Michael et al., 2005). Some suggest that the experience of intrusive memories following trauma is adaptive as it may encourage communication, reappraisal of the trauma and the accessing of social support (O'Donnell et al., 2007; Shalev & Rogel-Fuchs, 1993).

The experience of intrusive memories may lie on a continuum from everyday non-distressing intrusions to highly distressing and debilitating psychopathological intrusions (Holmes, Brewin, & Hennessy, 2004). Intrusive memories occur in the general population with a suggested prevalence of between 1-5 intrusions per day (Berntsen, 1996; Mace, 2005). Involuntary memories in the general population are associated with, on average, less positive more unusual events and have a greater impact on mood and physiological arousal than voluntary memories (Berntsen & Hall, 2004). Intrusions are also common in other psychological

disorders such as social phobia, depression and psychosis but differ in terms of their theme (Brewin et al., 2010). Intrusions, usually of adverse social events, are seen in social phobia (Hackmann, Clark, & McManus, 2000; Hirsch et al., 2003); severity of intrusions about previous abuse is associated with depression severity (Kuyken & Brewin, 1994); and intrusions have been documented in psychosis (Morrison et al., 2002). Some suggest (e.g. Steel, Fowler, & Holmes, 2005) that trans-diagnostic cognitive information processing mechanisms may underlie intrusion formation.

1.1.3 Summary

PTSD is a common, distressing and debilitating disorder that has serious consequences for the individual and society. PTSD symptoms are common after trauma with some suggesting that they might be a sign of normal adaptation. Distressing intrusions are the hallmark feature of PTSD, play a crucial role in psychological theories of PTSD and have been shown to predict PTSD development.

1.2 Memory and PTSD: cognitive models

Recent theories of PTSD have placed memory processes at the heart of the disorder and two models will be reviewed here: Ehlers & Clark's (2000) cognitive model and dual representation theory (DRT; Brewin, Dalgleish, & Joseph, 1996; Brewin et al., 2010). Both highlight memory encoding processes as fundamental to aetiology and treatment (Holmes & Bourne, 2008). They emphasize the role of 'faulty information processing' at the time of a trauma due to extreme emotion, which leads to the laying down of a memory trace with strong sensory features yet lacking context and a coherent time code (Ehlers & Clark, 2000; Holmes & Bourne, 2008). The disorganised memory is easily triggered by similar sensory cues leading to intrusive memories, the hallmark feature of PTSD.

Changes to memory functioning and information processing have been consistently identified in PTSD (e.g. recall bias to trauma cues; Buckley, Blanchard, & Neill, 2000). Studies have shown that the person's memory for the traumatic event improves over time (Mechanic, Resick, & Griffin, 1998), can contain gaps and be disjointed (e.g. Foa, Molnar, & Cashman, 1995; Harvey & Bryant, 1999) and that the content of the memory may change (Schwarz, Kowalski, & Mcnally, 1993; Southwick et al., 1997). Ehlers, Hackmann & Michael (2004) clearly state that "theorists concur that re-experiencing symptoms are due to the way the trauma memories are encoded, organised and retrieved."

1.2.1 Dual representation theory (Brewin, Dalgleish, & Joseph, 1996; Brewin et al., 2010)

Dual representation theory (DRT; Brewin, Dalgleish, & Joseph, 1996) proposes that trauma memories are represented in a fundamentally different way rather than, according to other theories, that they are an ordinary memory with a different structure. DRT postulates that there are two memory systems with distinct neural bases which act in parallel but one may take priority at certain times, for example under stress. Brewin et al. (2010) revised DRT in order to attempt to further embed the theory in cognitive neuroscience.

Brewin et al. (2010) defines abstract, contextually bound, consciously accessible representations as 'C-reps' (previously verbally accessible memories) and low level sensation-based, involuntarily accessed memories with emotional and affective components as 'S-reps' (previously situationally accessible memories). C-reps are proposed to be associated with the projection of the ventral visual stream to the inferior temporal cortex and its connections to the medial temporal lobe structures, whilst S-reps are proposed to be linked with the projection of the dorsal visual stream to the superior parietal areas with its connections to the insula and amygdala. Brewin et al. (2010) suggest that encoding usually involves the creation of S-reps and C-reps with connections established between them.

C-reps are integrated with other autobiographical memories, have a time code and a context and are stored in a form allowing deliberate retrieval and manipulation. However, they can only encode information that is consciously attended to and so may be restricted at times of high stress. Therefore C-reps encoded at the time of trauma are flexible, experienced in the past and can be modified but are also vague and contain gaps. S-reps can encode information that received less conscious attention and so can contain information not encoded by C-reps. S-reps are more extensive, lower level representations of the experience, such as sights and sounds as well as physiological responses (e.g. heart rate changes). S-reps lack verbal and contextual information (including time code) and are triggered by situational reminders of the trauma. S-reps are not verbally accessible and are poorly integrated in autobiographical memory. Therefore, S-reps are highly perceptual, triggered involuntarily and are experienced as if occurring in the present.

Brewin et al. (2010) propose that under extreme stress, attention is narrowed and hippocampal function is reduced, resulting in relatively stronger S-reps without the usual

integration with C-reps and so lack top-down control and context. This results in detailed and emotionally laden re-experiencing symptoms that are difficult to communicate with others and update with additional knowledge. Re-experiencing symptoms are proposed to be adaptive initially as they allow large amounts of information, potentially important to future survival, to be retained and re-processed in greater depth once the threat has passed. However, if there is inadequate integration of the S-reps and C-reps due to maladaptive strategies, such as behavioural and cognitive avoidance, then flashbacks remain persistent.

For example, extreme stress experienced during a traumatic road traffic accident (RTA) might narrow the person's attention and prevent them from processing the meaning and additional contextual details. S-reps might include highly detailed sensory aspects of the RTA (e.g. the sounds and smells associated with braking) and the emotions and physiological changes occurring during the RTA (e.g. fear and a racing heart). In contrast, the C-reps might encode details such as where and when (i.e. in the past) the RTA occurred. However, due to attentional narrowing the C-reps are vague and contain gaps and so cannot inhibit the S-reps, which are experienced as flashbacks. If, following the RTA, the person avoids thinking about what happened then the representation may not be sufficiently integrated and re-experiencing symptoms persist.

In terms of treatment, a key implication is the contextualisation of the S-reps and C-reps and the creation of memories that can be retrieved preferentially. Brewin et al. (2010) highlight the competition retrieval hypothesis where emotions and behaviour are under the control of memory representations that compete for retrieval. Negative representations cannot be directly changed but rather treatment aims to create and/or strengthen highly accessible and memorable positive representations. Recovery occurs when the positive representations are retrieved more consistently than the negative representations. Memory representations themselves are not fundamentally changed and so, given the right combination of cues, the trauma memories may be triggered in the future.

Brewin et al. (2010) highlight that therapeutic techniques, such as exposure and imagery rescripting, can facilitate contextualisation and integration of the S-reps. They propose that exposure treatments are effective as they facilitate the direction of attention to the S-reps allowing parts of them to be transferred into elaborated C-reps. In imagery rescripting, they postulate that when the intrusive distressing image is retrieved the corresponding C-rep and S-

rep are activated allowing integration and contextualisation of the S-rep. Recovery involves creating a memorable C-rep that is retrieved preferentially to the original representations. The DRT model places a greater emphasis on memory representations of the trauma than on cognitive appraisals.

1.2.2 Ehlers & Clark's (2000) cognitive model

Ehlers & Clark's (2000) cognitive model (see Figure 1) is considered to be the most detailed account of the maintenance and treatment of PTSD (Brewin & Holmes, 2003), with research evidence strongly supporting various aspects of the model. Ehlers & Clark (2000) propose that PTSD symptoms arise when the trauma is processed in a way that results in a strong sense of current threat despite the trauma being in the past. They identify two factors that lead to this sense of current threat: (1) Idiosyncratic misappraisals concerning the trauma and/or its sequalae and (2) the nature of the trauma memory. This current sense of threat results in a third factor: (3) maladaptive behavioural strategies and cognitive processing styles, that act to further maintain the disorder.

Misappraisals of the trauma and/or sequalae may include over-generalising threat (e.g. "the world is dangerous"), negative appraisals of symptoms (e.g. "I am going mad"), other peoples' reactions ("people will judge me") and the future (e.g. "I am disfigured and no-one will love me"). The trauma memory is proposed to be disorganised and insufficiently integrated with other autobiographical information, leading to poor intentional recall and distressing involuntary intrusions that convey a sense of current threat. The disorganised trauma memory is proposed to arise from 'faulty information processing' at the time of the trauma resulting in the memory lacking context, a coherent time code and abstract information (e.g. related autobiographical information) (Ehlers & Clark, 2000). Misappraisals and the disorganised trauma memory lead to a sense of current threat, including re-experiencing and hyper-arousal symptoms. The person seeks to control or manage their PTSD symptoms through unhelpful cognitive and behavioural strategies, for example avoidance and thought suppression. These strategies, used to attempt to reduce anxiety in the short term, act to maintain the sense of current threat by preventing change in the memory and the misappraisals.

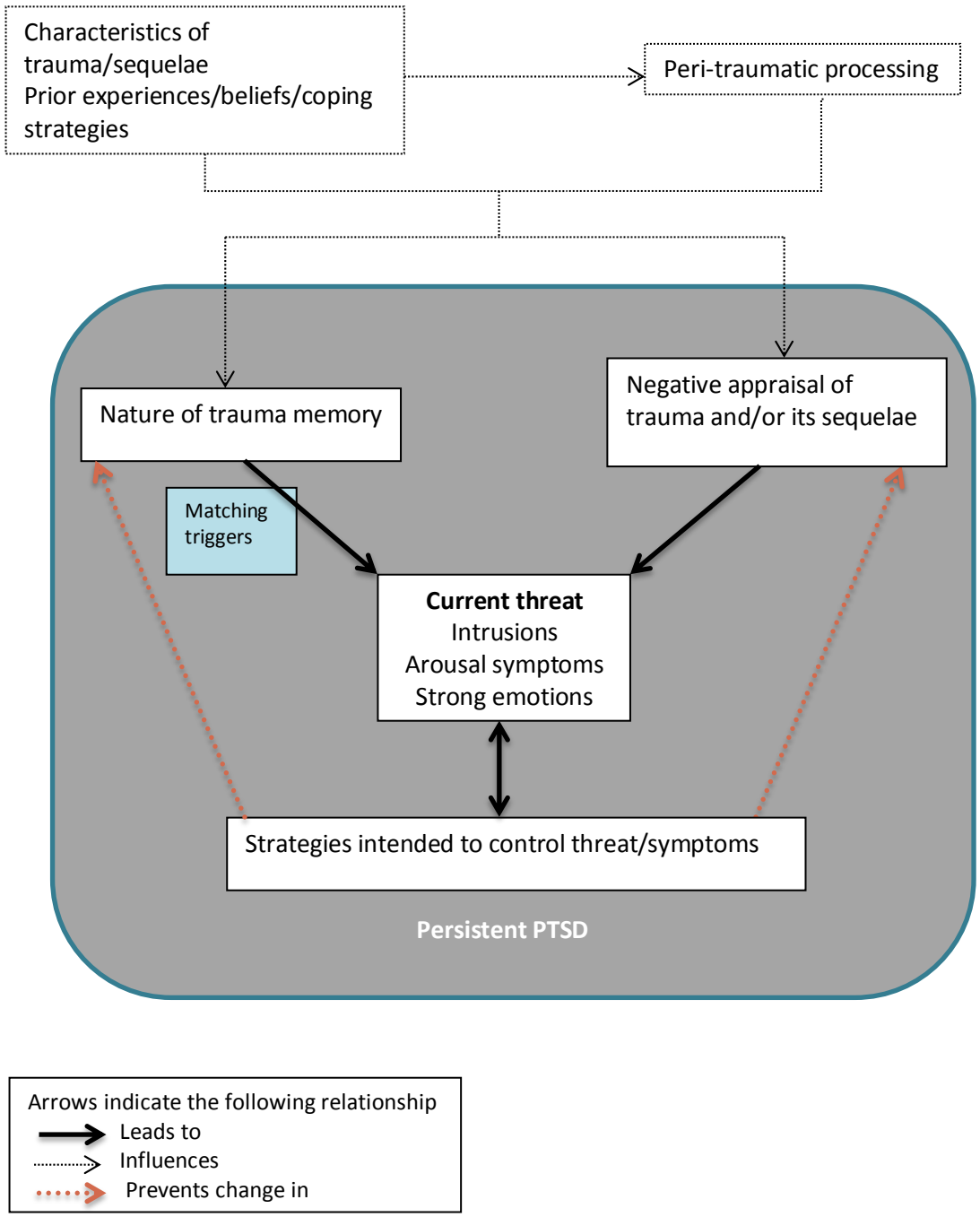
Ehlers & Clark (2000) propose two peri-traumatic processing styles: conceptual and data-driven processing. Conceptual processing, where the focus is on the meaning and context of the trauma, leads to the integration of the memory with autobiographical information. Data-

driven processing, where the focus is on sensory impressions such as sounds at the time of the trauma, leads to a memory which is difficult to retrieve intentionally. A shift away from conceptual processing at the time of trauma results in a distressing memory that is rich in sensory detail and experienced as if it is occurring in the present.

Ehlers & Clark (2000) also propose that, due to data-driven processing at the time of trauma, people with PTSD have stronger perceptual priming (where people are more likely to identify an object when previously exposed to it) and stronger associative learning (where neutral stimuli are associated with features of the trauma e.g. fear). These processes, combined with reduced resources to encode stimuli at the time of trauma, explain why re-experiencing symptoms are triggered easily and to a wide range of vaguely similar cues. Preferential identification of cues linked to the trauma (via perceptual priming) and a strong emotional response to signal potential threat (via associative learning) are proposed to be adaptive shortly after the trauma as they allow the individual to re-evaluate the safety of their environment (Ehlers, Hackmann & Michael, 2004). Usually re-experiencing symptoms decline as the memory is processed and as the person notices that triggers for their symptoms are not signalling threat. This process can fail when the person does not update the memory with new information or learn that the triggers do not signal present danger.

There is good evidence for this model for both adults and children, including retrospective and prospective studies. These have shown that the development and maintenance of PTSD is associated with processing the sensory details of the event rather than its meaning, a disorganised trauma memory, appraisals about the trauma and maladaptive coping styles (Bryant & Guthrie, 2007; Clohessy & Ehlers, 1999; Dunmore, Clark, & Ehlers, 1999; Ehlers et al., 2010; Ehlers, Mayou, & Bryant, 2003; Halligan et al., 2003; Mayou, Ehlers, & Bryant, 2002; Murray, Ehlers, & Mayou, 2002; Salmond et al., 2010). This model has clear implications for treatment including identifying and challenging idiosyncratic dysfunctional appraisals, elaborating the trauma memory and reducing maladaptive behaviours and cognitive strategies (e.g. Ehlers & Clark, 2008).

Figure 1: Ehlers & Clark (2000) cognitive model of PTSD



1.2.3 Summary

Key cognitive models have highlighted the fundamental role of memory processes in the development and maintenance of PTSD. Both theories highlight that a shift in information processing (for example from verbal-conceptual to sensory-perceptual) can occur under high stress and that this leads to the encoding of a memory that lacks sufficient integration with

other autobiographical memories. Both theories highlight the importance of contextualising and integrating the trauma memory.

1.3 Fear conditioning

Fear conditioning has been studied extensively in animals and has been extended to humans (for review see Delgado, Olsson, & Phelps, 2006). Fear conditioning is a process in which a neutral stimulus (conditioned stimulus; CS, e.g. a tone) gains the ability to evoke fear following repeated pairing with an aversive stimulus (unconditioned stimulus; US, e.g. a shock). This is because the CS+ (CS paired with the US) becomes a signal for US onset by activating the memory representation of the US. In general, fear conditioning is an adaptive and highly important form of learning. However, it may also be at the heart of the pathogenic mechanisms leading to anxiety disorders and has been implicated in theories for anxiety disorders for at least 80 years (e.g. Pavlov, 1927; Watson & Rayner, 1920). A meta-analysis (Lissek et al., 2005) found that people with anxiety disorders display a modest increase in acquiring fear learning and in conditioned responding to CS+ during extinction compared to people without anxiety disorders.

PTSD can be viewed as a lack of recovery from common post-traumatic psychological and physiological reactions. The majority of people (74% of women and 81% of men) experience a traumatic event in their lifetime (Breslau et al., 1998; Stein et al., 1997), almost all of these will initially develop PTSD symptoms (e.g. Rothbaum et al., 1992) and most will recover over time (Breslau et al., 1991; Kessler et al., 1995) with approximately 9% going on to develop PTSD (Breslau et al., 1998). Some suggest that re-experiencing and arousal symptoms are conditioned emotional responses resulting from classical conditioning and that the development of PTSD represents a lack of recovery due to an inability to extinguish the conditioned fear response (e.g. Rothbaum & Davis, 2003).

1.3.1 Conditioning theory of PTSD

Conditioning theory has been used to describe the development of PTSD since it was first included in the DSM in 1980 (Foa, Steketee, & Rothbaum, 1989; Foa, Zinbarg, & Rothbaum, 1992; Rothbaum & Davis, 2003). Researchers have proposed that Mowrer's (1960) two-factor learning theory can explain PTSD via classical and instrumental learning processes. In a conditioning model of PTSD, the trauma acts as the US and neutral stimuli (CS) present at the time of the trauma become fear-eliciting (i.e. provoke the conditioned response, CR) through

classical conditioning, consistent with the first stage of Mowrer's (1960) theory. Stimulus generalisation and higher order conditioning are proposed to then result in a large number of cues eliciting fear (e.g. Foa, Steketee, & Rothbaum, 1989). It would usually be expected that repeated exposure to the cues (CS+s) without US reinforcement would result in the extinction of the fear response. Conditioning theorists argue that no extinction occurs due to the person's reactions to the stimuli e.g. attempts to avoid the triggers and suppress memories. This is consistent with Mowrer's (1960) second stage, proposing that learned responses (e.g. avoidance) to the CS+ are developed to decrease anxiety and fear associated with the CS+. The person's avoidance of the CS+s enables a short-term reduction in fear and so is positively reinforced but maintains PTSD in the long term.

Conditioning theories offer a promising explanation of some of the central features of PTSD including why a wide range of previously neutral stimuli are capable of provoking physiological and emotional arousal and the role of avoidance in maintaining PTSD. Consistent with conditioning theory, evidence suggests that people with PTSD show heightened physiological activation to stimuli similar to those present at the time of trauma (Orr et al., 1997, 1998; Pitman et al., 1987) and people with PTSD may be more conditionable and more resistant to extinction than those without (Orr et al., 2000; Rothbaum et al., 2001). However, whether these differences result from PTSD or are pre-trauma vulnerability factors is unclear. Conditioning theory does fail to explain some important research findings such as the impact of coping strategies and appraisals (e.g. *perceived* threat is a better predictor of PTSD than *actual* threat; Foa, Steketee, & Rothbaum, 1989; Sales, Baum, & Shore, 1984) and researchers agree that the complexity of PTSD cannot be entirely explained by learning theory (e.g. Rothbaum & Davis, 2003).

1.3.2 Strategies to reduce the conditioned fear response

Fear conditioning can become a source of pathology when the person continues to anxiously react to the presence of a CS without the CS/US contingency being present. This evokes the question of how to weaken the CS/US association and three paradigms are reviewed: extinction, blockage of consolidation/reconsolidation and US devaluation. Unfortunately, reconsolidation blockage tends to require toxic drugs and extinction appears not to be permanent (Monfils et al., 2009). US devaluation, whilst not extensively researched, may have the advantage of acting directly on the US representation.

Extinction

In fear extinction, the fear response is reduced by learning that a previously threatening stimulus (CS+) no longer signals danger. The term extinction can refer to both the procedure used (in the laboratory this involves the repeated presentation of the CS+ in the absence of the US) and the outcome (i.e. the reduction in strength of the behavior to the CS+) (Bouton, 2007). Research suggests that fear extinction in humans depends on the same neural circuitry as other species, relying on prefrontal inhibitory mechanisms to regulate amygdala driven fear expression (Hartley & Phelps, 2010). However, extinction is not permanent and re-emergence of the original fear memory can occur. Re-emergence is commonly thought to happen under three general conditions: *renewal* occurring when the CS+ is presented outside of the extinction context (Bouton & Bolles, 1979), *reinstatement* occurring when the US is administered unexpectedly and *spontaneous recovery* occurring when a substantial amount of time has passed (e.g. Rothbaum & Davis, 2003). These processes imply that extinction involves the creation of new memories (inhibitory stimulus association CS+/noUS) that compete with the original fear memory (CS+/US) rather than changing the memory directly (Mark E Bouton, 2002, 2004). This indicates that extinction is fragile and can be disrupted by stress, time and a change in context.

In PTSD, a failure to consolidate or retrieve extinction learning may explain why people continue to re-experience fear spontaneously or in response to cues (e.g. Rauch, Shin, & Phelps, 2006). Extinction-based exposure therapies are used in the treatment of anxiety disorders and differences in individuals' extinction learning offers a potential mechanism to identify those at risk of developing anxiety disorders (Hartley & Phelps, 2010; Lommen et al., 2013). Not everyone benefits from exposure treatment (Ehlers et al., 1998) and deterioration following therapy can occur. For example, 40% of people receiving prolonged exposure still met diagnostic criteria following treatment (Foa et al., 1999) and Macklin et al. (2000) showed clinical deterioration following Eye Movement Desensitization and Reprocessing (EMDR) therapy over a five-year follow-up period. The return of fear following exposure is well documented (e.g. Craske et al., 2008; Richard & Lauterbach, 2007) and this may be due to the failure of the new extinction memory to inhibit the original fear memory. Whilst research into preventing the return of fear is important, the most effective method may be to prevent the consolidation of these fear associations in PTSD initially (Kearns et al., 2012).

Consolidation and reconsolidation

When fearful memories are initially formed they are labile and vulnerable to change but steadily become less so as they are consolidated via protein synthesis (Mcgaugh, 2000; Squire & Davis, 1981). The consolidation window is approximately six hours in rats (Nader, Schafe, & Le Doux, 2000) and it is suggested that memories are malleable for a similar period in humans (Holmes et al., 2009; Schiller et al., 2010; Walker, Brakefield, & Hobson, 2003). During this consolidation period, the formation of the memory can be disrupted and it was thought that, after consolidation, the memory was permanent (Hartley & Phelps, 2010). However, research has shown that during reconsolidation, a retrieved memory returns to this labile state and requires further protein synthesis to be stabilised. So, during consolidation and reconsolidation, there is an opportunity for the memory to be altered, updated or enhanced (e.g. Monfils et al., 2009; Nader, Schafe, & Le Doux, 2000).

Therefore, in the case of fear conditioning, consolidation and reconsolidation may act as an opportunity to alter the emotional impact of a fear conditioned stimulus. There is good evidence from non-human-animal models that targeting consolidation and reconsolidation using pharmacological manipulations can extinguish the conditioned fear response. These have used the Pavlovian fear conditioning paradigm and mostly used protein-synthesis inhibitors which are not safe for use in humans (e.g. Alberini, 2005; Doyère et al., 2007; Lee, Milton, & Everitt, 2006).

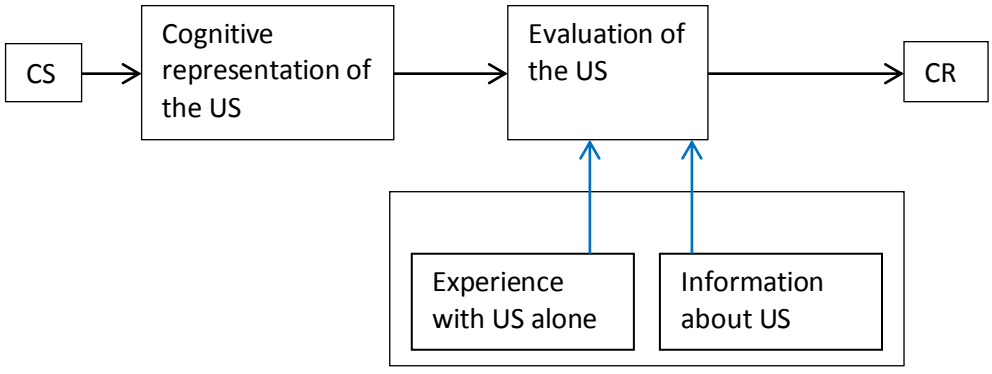
However, it has been illustrated that a non-invasive extinction procedure within either the consolidation or reconsolidation window can be used to re-write the fear memory and so prevent the return of fear in rats (Monfils et al., 2009; Myers, Ressler, & Davis, 2006). Little or no reinstatement, renewal or spontaneous recovery was seen in rats extinguished shortly after conditioning, compared to moderate to strong return of fear in rats extinguished 24 hours or more later. This has been replicated in humans using differential fear conditioning paradigms; studies illustrated that by extinguishing the fear memory during consolidation (Norrholm et al., 2008) or reconsolidation (Schiller et al., 2010), the spontaneous return of conditioned fear could be reduced. This was in contrast to the fear response being intact if the memory was updated outside the consolidation/reconsolidation window. The implication is that during consolidation or reconsolidation, the CS/US trace is labile and can be re-written to include the CS/noUS learning before storage. This creates an alternative memory trace, rather than a competing trace which is vulnerable to the return of fear. Early extinction may, therefore, offer

a mechanism to re-write fear memories and protect against the long-term psychological and physiological effects of traumatic fear memories. However, mixed results have been reported (Kindt & Soeter, 2011; Soeter & Kindt, 2011) and further investigation is warranted.

US devaluation

US devaluation is where the mental representation of the US is changed, for example through information or experience with the US. Devaluation can occur even without direct exposure to the US and results in changes to the CR when the CS+ is presented (e.g. Field, 2006; Dibbets, Poort, & Arntz, 2012). Conditioning theory suggests that the CS+ does not directly produce a CR but rather triggers a representation of the US and that the evaluation of this US mediates the CR (e.g. Davey, 1989) (see Figure 2). Therefore, the strength of the CR depends on the evaluation of the US and so altering this evaluation will modulate the CR's strength, independent of the CS/US contingency (Davey, 1989). For example, Rescorla (1973) demonstrated that, following CS/US pairing, if rats are habituated to the US then they show less fear to the CS+ compared to controls. The advantage of US devaluation, compared to extinction, is that it acts directly on the US representation and so may be less vulnerable to the return of fear and more easily generalisable. Therefore US devaluation could provide a more fundamental way to change the conditioned fear response compared to extinction procedures (Arntz, 2011).

Figure 2: US evaluation in modulating the CR (based on Davey, 1989)



Arntz et al. (e.g. 2011) propose that imagery rescripting (IR) may act through US devaluation. IR has been successfully applied to treating a number of anxiety disorders (Holmes, Arntz, & Smucker, 2007 for review; Hunt & Fenton, 2007; Wild, Hackmann, & Clark, 2007) including PTSD (Arntz, Tiesema, & Kindt, 2007). IR involves activation of the distressing memory and then updating/rescripting of the memory with neutral or positive information. Two recent

analogue studies illustrated that adding imagery rescripting could reduce intrusion frequency (Hagenaars & Arntz, 2012) and the conditioned fear response at renewal (using US expectancy ratings; Dibbets, Poort, & Arntz, 2012). Dibbets, Poort, & Arntz (2012) illustrated that adding US devaluation in extinction training reduced renewal of the fear response more than extinction alone. Interestingly, Dibbets, Poort, & Arntz (2012) indicated that IR may lead to slower extinction of the CS+, as measured by expectancy ratings, compared to groups that received extinction only and suggest this may be due to mental rehearsal of the CS/US relationship. However, Dibbets, Poort, & Arntz (2012) failed to show extinction or renewal of the conditioned fear response as measured by skin conductance response (SCR).

1.3.3 Summary

Conditioning theory provides a powerful account of how PTSD symptomatology may develop, including the variety of trauma-cues that elicit physiological arousal and the role of avoidance in maintaining PTSD. In terms of clinical work, it is important to consider that extinction can be fragile and can be disrupted by the passage of time, a shift in context or stress. Current psychological therapies for anxiety disorders use exposure to extinguish the conditioned fear response (Foa, Franklin, & Moser, 2002; Rauch, Shin, & Phelps, 2006) and potential for relapse is highlighted by studies illustrating that the extinguished fear response can return (e.g. Rothbaum & Davis, 2003). US devaluation and extinction during consolidation or reconsolidation may result in a more fundamental change to the fear memory and lead to the lasting loss of the fear response. Therefore, increased insight into how these mechanisms operate is crucial for optimising the efficiency of psychological interventions, especially the processes enabling the “unlearning” of behavioural patterns. However, these theories lack a cognitive component and cannot explain some importance research findings, such as the impact of beliefs on the development of PTSD.

1.4 Predictors of PTSD

In order to identify those people at risk of developing PTSD and develop effective prevention and early intervention programmes, it is essential to understand what factors make people more likely to develop PTSD. Studies have identified a number of individual risk factors for the development, severity and duration of PTSD including demographic, psychological and biological vulnerability factors. These can operate pre-trauma (e.g. psychiatric history), during the trauma (e.g. peri-traumatic dissociation) and post-trauma (e.g. lack of social support). Differences in psychophysiology and extinction learning have also been identified as risk

factors, with psychophysiological assessment identified as a potential mechanism to identify those at higher risk.

1.4.1 Demographic and cognitive variables

Various pre-trauma demographic variables and psychological factors have been identified as risk factors for developing PTSD. Demographic risk factors include female gender (Kessler et al., 1995; Perkonigg et al., 2000), previous trauma and lower social class (Perkonigg et al., 2000). Psychological risk factors include having an avoidant coping style (Bryant & Harvey, 1995), neuroticism (Breslau et al., 1991) and a depressive rumination style (Nolen-Hoeksema & Morrow, 1991). Prospective studies assessing appraisals prior to trauma illustrated that negative appraisals about ability to cope predicted PTSD four years later in fire-fighters (Bryant & Guthrie, 2007) and positive world assumptions are associated with lower rates of PTSD in police officers (Yuan et al., 2011). Poor cognitive functioning pre-trauma has been shown to predict PTSD in young adults exposed to natural disaster (Parslow & Jorm, 2007) and active Army personnel (Marx et al., 2009). However, different domains of cognitive functioning predicted PTSD in each of these two studies.

Features of the trauma itself and peri-traumatic processes increase risk of PTSD development, with psychological models of PTSD (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000) highlighting a central role of cognitive peri-traumatic processes. Aspects of the trauma itself associated with PTSD development include how long it lasts, its predictability and the type of trauma (Breslau et al., 1991; Ehlers & Clark, 2000; Kessler et al., 1995). Peri-traumatic processes including high anxiety and distress (e.g. Rothbaum et al., 1992) and cognitive factors such as mental defeat (Dunmore, Clark, & Ehlers, 1999), data-driven processing (Ehlers, Mayou, & Bryant, 2003; Ehlers et al., 2010; Halligan et al., 2003; Murray, Ehlers, & Mayou, 2002) and dissociation (e.g. Ehlers, Mayou, & Bryant, 1998; Weiss et al., 1995) are linked with PTSD development.

Post-trauma negative appraisals, sleep difficulties and unhelpful cognitive and behavioural coping styles have been investigated as predictors of PTSD. Onset, persistence and severity of PTSD are predicted by negative appraisals of the trauma and its sequalae, dysfunctional strategies (e.g. avoidance) and changes to the person's global beliefs (e.g. Bryant & Guthrie, 2007; Clohessy & Ehlers, 1999; Dunmore, Clark, & Ehlers, 1999; Ehlers & Steil, 1995). Sleep disturbance one month after trauma predicts PTSD development 12 months later (Koren et al.,

2002) and interventions targeting sleep improve PTSD symptomatology (Krakow et al., 2001). Efforts to suppress trauma memories and associated emotion, high avoidance and rumination have been highlighted as unhelpful coping strategies (Clohessy & Ehlers, 1999; Joseph & Williams, 1997; Laposa & Rector, 2012; Lawrence, Fauerbach, & Munster, 1996; Nolen-Hoeksema & Morrow, 1991). Disclosure, use of social support and attempts to understand the trauma have been indicated as helpful coping strategies (e.g. Jones & Barlow, 1990; Silver, Boon, & Stones, 1983).

Meta-analyses have attempted to bring together the findings to provide a coherent picture of risk factors. Meta-analyses indicate that peri and post-traumatic factors are better predictors of PTSD symptoms than pre-trauma factors such as adjustment, family psychiatric history or previous trauma (Brewin, Andrews, & Valentine, 2000; Ozer et al., 2008). Brewin, Andrews, & Valentine (2000) highlight significant moderator effects of sample size, design and measures and emphasise the need to take account of the dynamic interplay between pre, peri and post traumatic factors.

1.4.2 Biological vulnerability factors

Genetic and epigenetic factors, especially those linked to the stress-response gene pathways, may be risk factors for PTSD development. Studies have indicated genetic contributions to PTSD vulnerability (e.g. Koenen, Nugent, & Amstadter, 2008; Nemeroff et al., 2006) with twin studies indicating genetic influences on the likelihood of trauma exposure, development of PTSD and comorbidity (Koenen, Nugent & Amstadter, 2008). Studies also suggest that some gene variants (5-HTT, COMT, FKBP5) may modify both pharmacological and behavioural PTSD treatment response (e.g. Bomyea, Risbrough, & Lang, 2012).

Varying neuroendocrine responses to stress, for example pre-trauma hypothalamic-pituitary-adrenal (HPA) axis functioning, has also been linked to PTSD development (Bomyea et al., 2012). Studies suggest that low cortisol responses immediately following trauma lead to increased risk of PTSD development (Delahanty, Raimonde, & Spoonster, 2000; McFarlane et al., 2011). Some suggest that changes in neuroendocrine functioning during trauma leads to "superconditioning" and "overconsolidation" (Pitman, 1989) and that excess glucocorticoids may affect hippocampal function impacting on emotional regulation (Acheson, Gresack, & Risbrough, 2012; Conrad, 2008). Brewin et al. (2010) associates impaired hippocampal functioning with the creation of relatively stronger S-reps leading to re-experiencing

symptoms. The exact mechanisms by which genes and HPA axis functioning impact on PTSD development is currently unclear (for review see Bomyea, Risbrough, & Lang, 2012).

1.4.3 Predicting PTSD using psychophysiology

Hyper-arousal, and more specifically "physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event", is a characteristic feature of PTSD (APA, 1994). Clinical practice relies primarily on self-report to determine PTSD diagnosis and it is proposed that clinical and theoretical understanding might be enhanced by psychophysiological assessment (Orr & Roth, 2000). Studies have shown greater physiological reactivity to trauma-related cues in people with PTSD (Shalev & Rogel-Fuchs, 1993) and that PTSD severity may moderate responsivity to these cues (Keane et al., 1998; Orr et al., 1998).

There is evidence that changes in psychophysiology can predict development and persistence of PTSD as well as treatment outcome but the relationship is not straightforward. Increased (Shalev et al., 1998) and decreased (Blanchard et al., 2002) heart rate directly following trauma have both been linked with PTSD development and experimental studies using trauma films matched a decrease in peri-traumatic heart rate with increased intrusions (Holmes, Brewin, & Hennessy, 2004). In terms of treatment outcome, evidence suggests that early elevations in psychophysiology during imaginal flooding predict improvements in intrusions (Pitman et al., 1996) and that reductions in psychophysiological responses during trauma-related imagery occurs following treatment (Lindauer et al., 2006) and is associated with better adjustment (Boudewyns & Hyer, 1990).

Acquisition and extinction learning as a predictor for PTSD

Individual differences in fear conditioning have been proposed as a predictor of PTSD. Increased acquisition of fear learning has been demonstrated in people with anxiety disorders (Lissek et al., 2005) and people with PTSD show delayed fear extinction, both in terms of physiological measures and US expectancy ratings, compared to controls with and without trauma exposure (Orr & Roth, 2000; Peri et al., 2000; Blechert et al., 2007). Preliminary evidence that extinction learning may be a pre-trauma vulnerability factor was demonstrated by Guthrie & Bryant (2006) who showed that reduced extinction learning in fire-fighters predicted PTSD symptoms up to two years later. A large prospective study illustrated that reduced extinction learning in soldiers before deployment to Afghanistan predicted PTSD

symptom severity (Lommen et al., 2013) more strongly than neuroticism, pre-deployment stress and exposure to stressors on deployment.

1.4.4 Summary

Whilst a range of psychological and demographic factors are linked to the development of intrusive memories and PTSD, it appears that peri and post traumatic factors may be more instrumental. Whilst exact mechanisms remain unclear, it has been suggested that changes to endocrine functioning during trauma may lead to memories being over-consolidated and so highly resistant to extinction. Psychophysiology assessment of fear conditioning offers some promising results for predicting those more vulnerable to PTSD development.

A major limitation for this research is its reliance on retrospective self-report due to a general difficulty accurately recalling past emotional states, especially during traumatic experiences (Candel & Merckelbach, 2004). Causality of PTSD development cannot be concluded from these studies and contamination with current post-traumatic stress cannot be excluded. Prospective longitudinal studies have mostly been confined to at-risk occupational groups and how much these findings can be generalised is unclear. Therefore, experimental studies are essential to build a better understanding of the causal relationship between risk factors and PTSD development.

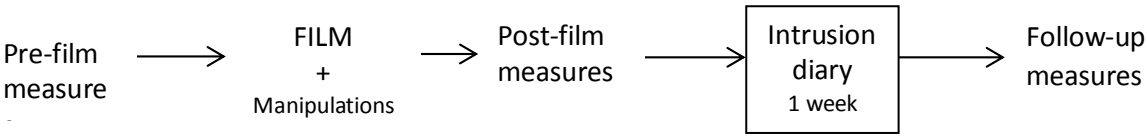
1.5 Analogue studies: the trauma film paradigm

Prospective studies are essential to examine what factors lead to PTSD development and persistence, yet it is clearly unethical to expose people to real trauma. The trauma film paradigm provides an experimental tool to prospectively induce and manipulate affect-laden memories in non-clinical controls in the laboratory. The trauma film paradigm was introduced by Lazarus et al. (1965) and further developed by Horowitz (1975). This paradigm is capable of inducing intrusions and other analogue PTSD symptoms (e.g. fear, avoidance, and arousal) in non-clinical participants (Laposa & Alden, 2006; Halligan, Clark, & Ehlers, 2002) implying that it is a useful analogue to real-life trauma (Holmes & Bourne, 2008). Intrusions are a precursor and the hallmark symptom of PTSD and intrusion frequency is a common outcome measure for the trauma film paradigm (Holmes & Bourne, 2008).

In the trauma film paradigm, participants are exposed to short films containing distressing events (e.g. road traffic accidents) and their responses are measured (e.g. frequency of

intrusions, arousal) under different conditions. Holmes & Bourne (2008) suggest the following methodology:

Figure 3: Trauma paradigm methodology



Early studies illustrated that trauma films can produce marked physiological stress responses and that the stress response can be experimentally manipulated (e.g. Lazarus et al., 1965). Whilst trauma films may not lead to the intensity of emotional reaction associated with experiencing a trauma, Holmes & Bourne (2008) suggest that intrusions may develop on a continuum of stressfulness from viewing fictional distressing films, watching real-life trauma, viewing self-related trauma films to experiencing trauma in real life. Research using this paradigm has illustrated that intrusion frequency can be reduced or increased according to predicted manipulations, including cognitive coping strategies (Laposa & Alden, 2006) and introducing a competing task (Holmes, Brewin, & Hennessy, 2004; Holmes et al., 2009). Most research using the trauma film paradigm has concentrated on peri-traumatic processing.

1.5.1 Experimental studies: predictors of PTSD

Consistent with predictions from psychological models (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000), the trauma film paradigm has been used to investigate how changes in information processing affects intrusion frequency. Both models predict that a shift from verbal to visuospatial processing at encoding results in the development of re-experiencing symptoms. Therefore if verbal encoding is enhanced or visuospatial encoding is disrupted, re-experiencing symptoms should decrease and vice versa. It has been shown that disrupting verbal processing during encoding increases intrusion frequency and disrupting visuospatial processing during (Holmes, Brewin, & Hennessy, 2004; Stuart, Holmes, & Brewin, 2006) or after encoding (Deeprose et al., 2012; Holmes et al., 2009) decreases intrusion frequency compared to controls. Holmes et al. (2009) argue that disrupting visuospatial processing leads to poorer encoding of the sensory and perceptual details associated with intrusions, whilst disrupting verbal encoding leads to a reduced ability to suppress sensory memories resulting in more intrusions.

However, subsequent studies have failed to replicate the finding that verbal interference leads to an increase in intrusions (Krans, Naring, & Becker, 2009; Pearson & Sawyer, 2011). Krans, Naring, & Becker (2009) suggest that this may be because Holmes, Brewin, & Hennessy (2004) incorporated an introductory passage giving context to the films that may have activated verbal-conceptual processing. Holmes, Brewin, & Hennessy (2004) also predicted that enhancing verbal processing would reduce intrusion frequency but did not find a significant effect. Holmes & Bourne (2008) suggest this may be because participants used surface level descriptions rather than engaging with the emotional meaning of the films, which may be insufficient to shift processing enough towards verbal-conceptual processing.

Studies have investigated whether asking participants to generate a processing style is effective in reducing intrusions. One study found that asking participants to adopt a conceptual rather than a perceptual processing style made no difference to intrusion frequency but having a trait perceptual processing style predicted intrusion frequency, distress and avoidance (Halligan, Clark, & Ehlers, 2002). In a two-part study, Laposa & Alden (2006) interviewed emergency service workers to identify effective coping strategies and then asked healthy participants to implement these strategies whilst watching a film of a hospital emergency room. Participants in the coping group reported fewer intrusions than those in the control group. Adding to this, some studies have trained participants to process the films in a particular way. Schartau, Dalgleish, & Dunn (2009) illustrated that explicitly training individuals to generate functional appraisals (e.g. “seeing the bigger picture”, “bad things happen”) during or after trauma films led to decreases in psychophysiological responses and self-reported distress and horror. Schartau, Dalgleish, & Dunn (2009) also illustrated that appraisal practice can lead to fewer intrusions and avoidance of distressing autobiographical memories for participants with higher levels of negative affect. Woud et al. (2012) trained participants in positive or negative appraisals of their self-efficacy and emotional responses to the films. Those in the negative condition had more intrusions and higher scores on the Impact of Events Scale (Weiss & Marmar, 1997) compared to those in the positive condition.

The trauma film paradigm has also been used to investigate peri-traumatic dissociation and post-trauma factors such as rumination, thought suppression and worry. Studies have linked state (Holmes, Brewin, & Hennessy, 2004) and trait (Murray, Ehlers, & Mayou, 2002) dissociation and state rumination (Zetsche, Ehring, & Ehlers, 2009) with intrusion development. However, experimental studies attempting to induce peri-traumatic dissociation

(for example by hypnosis) and rumination have failed to show a significant effect on intrusion frequency (Hagenaars et al., 2008; Zetsche, Ehring, & Ehlers, 2009). Kindt, Van den Hout, & Buck (2005) have demonstrated that self-reported spontaneous peri-traumatic dissociation correlated with memory fragmentation and intrusion frequency over a four-hour period. Studies have illustrated that thought suppression (Davies & Clark, 1998a) and worry (Butler, Wells, & Dewick, 1995; Wells & Papageorgiou, 1995) following the films leads to higher intrusion frequency. Worry, rumination, thought suppression and dissociation may all be forms of cognitive avoidance which prevent emotional processing of the trauma, and studies have been mixed in their ability to experimentally manipulate these processes in the lab.

1.5.2 Summary

The trauma film paradigm offers a novel, effective and ethical approach for prospectively investigating causal factors linked to the development and maintenance of PTSD. Studies have highlighted the role of peri and post-traumatic processes in the development of intrusions. These studies have provided evidence that a shift towards visuospatial processing leads to a higher number of intrusions and that training in appraisal style can reduce intrusions. Whilst offering insight into PTSD development, these studies also highlight areas for early intervention and importantly suggest that prophylactic measures are possible.

1.6 Psychological interventions for chronic PTSD

NICE (2005) recommend Trauma-Focused Cognitive Behavioural therapy (TF-CBT), based on Ehlers & Clark's (2000) cognitive model, or Eye Movement Desensitization and Reprocessing (EMDR; Shapiro, 1989) for chronic PTSD. Meta-analyses (NICE, 2005; Bisson et al., 2007) concluded that TF-CBT and EMDR demonstrate clinically important benefits over waiting-list and other supportive treatments, with the strongest evidence for TF-CBT. TF-CBT can be effectively disseminated into routine clinical services (Gillespie et al., 2002) and has low drop-out rates suggesting that it is acceptable to patients (Ehlers et al., 2005).

A central aim of both EMDR and TF-CBT is to integrate the trauma memory more fully into autobiographical memory. The features of the trauma memory (e.g. lack of context and temporal order) indicate that people with PTSD need to reconstruct the event. People that recover spontaneously from PTSD symptoms presumably are able to reconstruct the experience through natural recovery by talking and thinking about the event. For those who continue to suffer from PTSD symptoms, reconstruction and elaboration of the trauma

memory is believed to be an essential treatment goal (Ehlers & Clark, 2000). Ehlers & Clark (2008) emphasise that the most effective treatments for PTSD are those focusing on the person's trauma memory and its meaning. There have been a number of ways developed to enable this reconstruction including prolonged exposure (Foa & Rothbaum, 2001) and cognitive updating (Ehlers & Clark, 2000).

1.6.1 Therapeutic tools: exposure

Foa and colleagues have highlighted the effectiveness of using systematic exposure as a tool for treating PTSD (Foa et al., 1991; Foa & Rothbaum, 2001). Exposure involves confronting anxiety-provoking trauma-related stimuli with the aim of habituating to these stimuli and so no longer finding them psychologically or physiologically arousing. Patients are usually asked to speak in the first person present tense about their trauma experience as if it was happening again (e.g. Richards & Rose, 1991). Exposure can be conducted in a number of ways including imaginally and in vivo (e.g. in places associated with the trauma but not currently dangerous). Exposure is based on learning theory i.e. that continued presentation of a CS+ without the US leads to extinction of the fear response. As is seen in extinction, exposure therapy is effective at reducing fear but fear can return (Craske, 1999; Macklin et al., 2000; Rachman & Lopatka, 1988; Richard & Lauterbach, 2007). It is postulated that extinction does not lead to unlearning of the CS/US association but rather produces a memory that competes for retrieval with the original fear memory.

1.6.2 Therapeutic tools: updating

A key difference between TF-CBT and other effective therapies are the techniques for actively incorporating updated information into the trauma memory. This is thought to be important to gain full benefit from therapy (Ehlers, Hackmann & Michael, 2004). In therapy, this involves three steps (e.g. Ehlers & Clark, 2008):

1. Identify the worst moments of the trauma that create the greatest distress and sense that they are occurring in the present ("hotspots") through imaginal reliving and discussion of intrusions.
2. Identify information that updates the trauma memory either by identifying characteristics of the event (e.g. the outcome) or by cognitive restructuring.
3. Actively incorporate the information (identified in step 2) into the hot spots (from step 1) using verbal and imagery techniques during reliving of parts or all of the trauma memory.

The relative effectiveness of exposure versus updating and whether combining the two (as in TF-CBT) is even more effective is contentious. Marks et al. (1998) illustrated that, at six-month follow-up, exposure and cognitive restructuring alone were similarly effective in treating chronic severe PTSD and combining the two did not produce better outcomes. CBT and exposure therapy have been shown to produce similar outcomes (Paunovic & Ost, 2001) but some studies have illustrated superiority of CBT over imaginal exposure at five-year follow-up (Tarrier & Sommerfield, 2004), despite finding no differences between the groups following treatment or at 12-month follow-up (Tarrier et al., 1999). How similar the active treatment mechanisms for these two techniques are is debated and it may be that their relative effectiveness changes depending on PTSD chronicity and severity. For example, Bryant et al. (2008) showed that exposure was more effective than cognitive restructuring in reducing PTSD symptoms in people with Acute Stress Disorder (ASD).

It is recognised that, following exposure, some patients may experience initial adverse reactions such as increased re-experiencing symptoms (e.g. Foa, Franklin, & Moser, 2002) and that clinicians may avoid using exposure therapies due to concerns about this (Ehlers & Clark, 2008). However, there is evidence to suggest that prolonged exposure does not result in increased drop-out rates or aversive reactions compared to cognitive restructuring (Bryant et al., 2008).

1.6.3 Summary

NICE guidelines recommend TF-CBT and EMDR for treating chronic PTSD and both aim to integrate the trauma memory more fully into autobiographical memory. Exposure and cognitive updating are central therapeutic tools in evidence-based PTSD treatments but their relative effectiveness is unclear.

1.7 Early psychological interventions

Due to the devastating impact of PTSD for the individual, large costs associated with it and the lack of spontaneous recovery in a substantial proportion of sufferers, there is an urgent need to develop effective interventions that can prevent the development of the disorder (e.g. Kearns et al., 2012) and reduce the duration of disability. There is also a drive for effective interventions for occupational groups frequently exposed to trauma (e.g. journalists in conflict zones, emergency service workers and military personnel) and for relief workers following

large-scale disasters. Despite the successful development of treatments for persistent PTSD, there is mixed evidence for early interventions; some one-time interventions may actually worsen recovery rates (i.e. critical incident stress debriefing; Van Emmerik et al., 2002) whilst others, especially those offering multiple sessions targeting at risk groups, may successfully prevent the development of PTSD. Whether the intervention is harmful, helpful or neutral may be associated with a number of factors including the timing of the intervention, the number of sessions and the person's risk status for developing PTSD.

'Psychological Debriefing' (PD), first developed in World War One, aims to aid coping and reduce distress by intervening in the immediate aftermath of a trauma with support, psycho-education and emotional expression. Systematic reviews have shown that single session PD is ineffective, potentially slows recovery and its implementation is not recommended (Cochrane review: Rose et al., 2002; NICE, 2005). Studies have found that people receiving PD had more physical health problems, travel anxiety, general psychiatric symptoms and were more likely to be symptomatic for PTSD than those who received no intervention (Mayou, Ehlers, & Hobbs, 2000; Bisson et al., 1997). A dismantling study by Sijbrandij et al. (2006) suggests that increased PTSD symptoms may result following emotional debriefing, as opposed to educational debriefing, in individuals with higher baseline hyper-arousal.

Other brief psychological interventions that have been developed include self-help and psycho-education, group-based interventions and stepped-care approaches. Studies using self-help and psycho-education have not produced significant results (Turpin, Downs, & Mason, 2005; Bugg et al., 2009; Scholes, Turpin, & Mason, 2007). However, there is preliminary support for group-based psycho-education targeting at-risk occupational groups, such as military personnel (Adler et al., 2009). Resnick et al. (1999) illustrated that a person's distress during forensic rape examination predicted psychopathology six weeks later and that this distress could be reduced by a video intervention providing information and coping strategies. Stepped-care approaches for acutely injured trauma patients, involving case management supplemented with CBT, psychopharmacology and motivational interviewing, have also been shown to reduce PTSD symptoms (Zatzick et al., 2004).

Cognitive behavioural techniques administered to people with ASD in the first month after trauma are the most effective early intervention so far (Rothbaum et al., 2012). They have been shown to reduce rates of PTSD and anxiety (Bryant et al., 1999; Foa, Zoellner, & Feeny,

2006) and NICE (2005) recommend TF-CBT is offered to people with severe post-traumatic stress symptoms in the first month following trauma. Studies that have compared exposure and cognitive therapy at early intervention have shown mixed results: Shalev et al. (2012) illustrated comparable results in reducing PTSD whilst Bryant et al. (2008) found that those receiving exposure were more likely to be in full remission at follow-up and achieve higher functioning than those receiving cognitive restructuring. Bryant et al. (1999) suggest that prolonged exposure may be the critical component in CBT to prevent ASD developing into PTSD. Whilst these studies all offer promising results for intervention weeks after trauma, immediate intervention following trauma exposure and so prevention of PTSD might be even more effective.

Pharmacological studies have suggested that there is a "window of opportunity" following trauma to prevent PTSD becoming chronic (Zohar et al., 2011). Glucocorticoid treatment following trauma exposure may be effective in reducing the conditioned fear response which is linked to the development of PTSD via enhanced consolidation (Jovanovic et al., 2011; Miller et al., 2011). Concerns about early pharmacological interventions include side effects and ethical concerns that pharmacological agents might lead to the suppression of voluntary human memory including, for example, how this might impact on a court case. Shalev et al. (2012) compared psychological and pharmacological interventions and found that CBT was effective in reducing PTSD post-treatment compared to waitlist controls and no difference between those receiving a selective serotonin reuptake inhibitor (SSRI) or a placebo pill. However, there were no differences in rates of PTSD between the groups at nine-month follow-up.

Analogue experiments using the trauma film paradigm (see previous section) highlight that a competing visuospatial task after trauma (Holmes et al., 2009) and training in appraisal style (Schartau, Dalgleish, & Dunn, 2009; Woud et al., 2012) can reduce intrusion frequency and PTSD symptoms. It has also been demonstrated that extinction training during consolidation reduces the conditioned fear response compared to those receiving extinction training after 72 hours (Norrholm et al., 2008). This implies that extinction occurring early after trauma may disrupt consolidation of the pathological trauma memory and so offer an early intervention mechanism to reduce risk of PTSD development (Kearns et al., 2012). Rothbaum et al. (2008, 2012) delivered a brief exposure-based intervention in the emergency department immediately following trauma and produced promising results: people receiving the intervention had lower levels of depression and post-traumatic stress reactions at follow-up.

This provides a potential avenue for non-invasive preventative interventions based on psychological theory and requires further investigation.

1.7.1 Summary

An enduring hope in mental health research is to be able to prevent psychopathology rather than treating symptoms following their onset. It is crucial to develop effective prevention and early intervention techniques to limit the hugely detrimental impact of PTSD and reduce risk for occupational groups frequently exposed to trauma. There has been mixed evidence for early interventions: single session 'psychological debriefing' has been shown to be ineffective and potentially harmful, whilst there is positive evidence that cognitive behavioural techniques administered shortly after trauma may reduce PTSD development. It has been suggested that there is a "window of opportunity" following trauma when consolidation of the memory trace can be disrupted, and preliminary evidence suggests that psychological intervention in this period may reduce psychopathology.

1.8. Summary of literature and rationale

The majority of people will experience a traumatic event in their lifetime and most will develop PTSD symptoms following trauma that typically extinguish over time. However, approximately 9% will go on to develop PTSD (Breslau et al., 1998). PTSD is a distressing and debilitating condition which has serious implications for the individual and for society. Involuntary highly emotive and intensely distressing intrusions about the trauma are the hallmark feature of PTSD. These intrusions can be exceptionally vivid and experienced as if the trauma is occurring again. Most people experience intrusions following trauma and high initial levels of intrusions have been shown to predict PTSD development (O'Donnell et al., 2007). Cognitive models of PTSD highlight the role of memory encoding at the time of trauma on the formation of re-experiencing symptoms (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000). A shift in information processing from verbal-conceptual to sensory-perceptual processing in times of extreme stress is postulated to lead to memories that are high in sensory detail, lack a coherent time code and context and cannot be inhibited by usual top-down processes. Re-experiencing symptoms and their associated distress may lead to other PTSD symptoms and maintain the disorder (e.g. Ehlers & Clark, 2000).

Conditioning theory complements and is included in some cognitive theories of PTSD. It provides an account of how PTSD may develop, including how trauma-cues can elicit physiological arousal and the role of avoidance in maintaining PTSD. Models investigating neuroendocrine functioning suggest that "superconditioning" may occur at the time of trauma resulting in memories that are more resistant to extinction (Pitman, 1989). Extinction, consolidation and US devaluation are important concepts when considering interventions for PTSD. Exposure-based therapies use extinction to reduce the conditioned fear response but extinction is not permanent and fear can return in time, due to a shift in context or stress. Extinction is proposed to lead to the creation of competing memory traces rather than directly changing the trauma memory itself. Consolidation and reconsolidation may represent "windows of opportunity" where trauma memories are malleable and encoding can be disrupted. This may allow the original fear memory to be directly altered and so prevent PTSD development. Whilst extinction creates a competing inhibitory stimulus association (CS/noUS) with the original fear memory, US devaluation changes the mental representation of the US to reduce the CR. So, US devaluation during consolidation may directly alter the US representation and so act as a powerful mechanism to reduce the conditioned fear response and prevent it from returning.

A variety of vulnerability factors are associated with PTSD development. Peri-traumatic and post-traumatic processes seem particularly important in understanding who develops PTSD and why. The trauma film paradigm offers a powerful tool to investigate causal factors in PTSD development. Excitingly, this paradigm has been used to show that changes in appraisal style (e.g. Schartau, Dalgleish, & Dunn, 2009) and a shift away from visuospatial processing (e.g. Holmes et al., 2009) leads to fewer intrusions, as predicted by cognitive models. Alongside this prediction, is that increasing verbal processing should result in fewer intrusions but studies have failed to show this. Holmes & Bourne (2008) suggest that this may be due to participants not engaging enough with the emotional meaning of the films.

Understanding the mechanisms that make psychological interventions effective and developing early interventions is essential to limit the devastating impact of PTSD. Exposure and updating are central therapeutic tools in evidence-based PTSD treatments but their relative effectiveness is unclear. There has been mixed evidence for early interventions but implementing cognitive behaviour techniques after trauma has gained support. There is some evidence to suggest that the critical component for these early interventions are exposure techniques rather than cognitive restructuring (Bryant et al., 2008).

Reducing the fear response is a central aim in therapies for PTSD. Research has shown that it is possible to condition the fear response in humans in the laboratory, that the fear response can return following extinction and, importantly, that it is possible to prevent it from returning (e.g. Schiller et al., 2008, 2010). There is also evidence that individual differences in fear extinction predicts PTSD development (Lommen et al., 2013). This study aims to investigate whether changing the meaning of the US during memory consolidation is effective in reducing the conditioned fear response and PTSD symptomatology and whether it is equivalent to or more effective than behavioural methods. It is proposed that updating the meaning of the films will verbally enhance the fear memory and cognitively devalue the US representation, leading to the more effective reduction of the conditioned fear response and fewer analogue PTSD symptoms than exposure alone. Therefore, this study is investigating the relative effects of frequently used therapeutic techniques, updating and exposure, on the conditioned fear response, intrusion development and PTSD symptomatology in order to inform early intervention and treatment development. It is essentially asking whether adding a cognitive component to a US devaluation process reduces the potency and durability of a distress-associated stimulus.

1.9 Aims

1. To investigate whether the conditioned fear response can be acquired using trauma film stimuli as the US and whether the reduction and re-acquisition of this response can be experimentally manipulated.
2. To investigate whether updating the meaning of the trauma films ('update' group) compared to further exposure to them ('exposure' group) or watching neutral films ('neutral' group) has an effect on (a) the reduction of the conditioned fear response according to SCR and subjective distress ratings and (b) intrusion frequency, intrusion distress and the development of associated PTSD symptoms in the week following the experiment.
3. To investigate whether individual differences in fear conditioning and maladaptive responses to memories of the films are associated with intrusion frequency, intrusion distress and PTSD symptomatology in the week following the experiment.

1.10 Experimental design

This research followed an experimental between-subjects design with four main stages: acquisition, US devaluation, re-acquisition and follow-up. The design is an adaptation of the one used by Schiller et al. (2010). In the acquisition phase, all participants underwent the same fear conditioning paradigm using trauma film stimuli as the US. Participants were then randomly allocated to one of three US devaluation groups: updating, exposure or neutral. The update group viewed the films again but was given additional verbal information about what happened to the protagonists of the films, the exposure group viewed the films again and the neutral group viewed non-traumatic films of related content. Following the US devaluation phase, the conditioned stimuli were re-paired with the US in the re-acquisition phase. After completing the experiment, participants were asked to complete an intrusions diary for one week and complete follow-up questionnaires one week later. The acquisition and US devaluation phases were carried out consecutively to ensure that US devaluation occurred within the consolidation window.

Questionnaires were administered before the acquisition stage and at one-week follow-up. The independent variable was the US devaluation group that participants were allocated to (with three levels: update, exposure and neutral). The dependent variables were:

1. The participant's physiological and subjective response to the CS+ (neutral stimulus paired with the US) and CS- (neutral stimulus not paired with the US) before and after each phase.
2. The number of and distress caused by intrusive images or thoughts of the films in the week following the experiment.
3. PTSD symptomatology at one-week follow-up, as measured by the Impact of Events Scale (Weiss & Marmar, 1997).

A pilot study was initially carried out to evaluate the feasibility of the study.

1.11 Research hypotheses

Hypothesis 1: Participants will produce the conditioned fear response when trauma film stimuli are used as the US. Conditioning is defined as having an SCR amplitude to the unreinforced CS+ that is greater than 0.02µs and that SCR and subjective distress ratings are greater to the CS+ than the CS-.

Hypothesis 2: (A) Participants in the update group will have the largest reduction in physiological response and subjective distress ratings following the US devaluation phase compared to those in the exposure and neutral groups. (B) Participants in the update group will have the smallest increase in conditioned response and distress ratings following the re-acquisition phase.

Hypothesis 3: Participants in the update group will experience fewer intrusions, be less distressed by them and have fewer PTSD symptoms in the week following the trauma paradigm than the exposure or neutral groups.

Hypothesis 4: Participants who have a smaller conditioned fear response following acquisition will experience fewer intrusions, be less distressed by them and report fewer PTSD symptoms in the week following the trauma paradigm.

Hypothesis 5: Participants who have a lower score on the Response to Memories questionnaire (R2M) will experience fewer intrusions, be less distressed by them and have fewer PTSD symptoms in the week following the trauma paradigm.

2. Methods

2.1 Power analysis and sample size

A power analysis was conducted using Cohen's power primer in order to estimate the sample size required. In order to estimate effect sizes, we used the results from a similar study by Schiller et al. (2010) which used a between-subject design with three groups to investigate how different reconsolidation conditions can alter the likelihood of the conditioned fear response returning. Schiller et al. (2010) found that the difference between the recovery of fear (re-extinction vs. extinction phases) across the three groups indicated a large effect size of d=1.02. Using a 2-way analysis of variance (ANOVA) with 3 groups design, Cohen's power primer indicated a sample size of n=21 per group with alpha set at 0.05 and power at 80% for a large effect. Therefore, power analysis indicated that a total sample size of 63 would have 80% power to detect significant differences in change scores between the three groups.

The piloting stage identified that some participants demonstrate poor electrodermal responsivity (as measured by skin conductance response, SCR). Researchers recommend deciding upon a minimum amplitude change in conductance to count as an elicited SCR (Dawson, Schell, & Filion, 2007). Consistent with previous research (Kindt & Soeter, 2011; Schiller et al., 2010; Schiller et al., 2008; Soeter & Kindt, 2011), a minimum response criteria of 0.02µs was used in this study. The power analysis is based on participants who show an SCR amplitude above 0.02µs. Therefore, the sample size was adjusted according to the number of participants meeting minimum response criteria during the acquisition phase. From the piloting stage, it was estimated that approximately 25% would fail to produce a SCR above 0.02µs. Therefore, the sample size was estimated to be 84, with approximately 21 of these predicted to show no response, leaving a sample of 63.

2.2 Participants

115 participants completed the experimental task: 28 males and 87 females with a mean age of 26.72 (Standard Deviation [SD] 7.842; range 18-56). 75 participants (21 males and 54 females) met minimum response criteria for SCR with a mean age of 24.88 (SD 5.337; range 18-51). Participants were recruited using circular emails (see Appendix 1) to staff and students

at Kings College London and on MindSearch, a database of healthy volunteers. Exclusion criteria included being under 18 years of age, having completed similar trauma studies, working mostly in Accident and Emergency services (A&E) or hospitals and having a clinically significant mental health problem as assessed by scoring above clinical cut-off on standardised measures of anxiety, depression and/or PTSD. Participants were asked to complete the screening questionnaires before attending the one hour session. Before attending the session, participants were randomly allocated to a US devaluation group (update, exposure or neutral). At the screening stage, 25 participants were excluded due to having clinically significant mental health problems and six participants were excluded for having previously completed a similar study or for having A&E experience.

2.3 Ethical considerations

In this study, the purpose of using the trauma film paradigm was to induce physiological arousal in the form of fear. Therefore, ethical considerations were essential to minimise distress. Holmes & Bourne (2008) list ethical safeguards that should be put in place, these include: exclusion of participants with mental health difficulties; clear information to participants about film content prior to participation; use of precautionary measures to deal with distressed participants (most commonly used is that the study is conducted under the guidance of a clinical psychologist); and provision of contact details to the participants after the study has ended in case they have any concerns. All of these safeguards were put in place for this study. In addition, we used established trauma films that have been administered in previous studies without producing harmful side effects. The content of the films was also similar to what may be witnessed in a television or news programme.

2.3.1 Ethical approval

The Psychiatry, Nursing and Midwifery Research Ethics Committee at Kings College London approved the original ethical application and modification following piloting (see Appendix 2 & 3).

2.4 Measures

Participants were asked to complete measures before beginning the experimental task, after the task and one week after completing the task. The measures completed before the task acted as screening tools in order to exclude participants that had clinically significant levels of

mental health problems, as well as to match the groups according to demographic factors, levels of anxiety, PTSD and depression and trauma history. The measures that were used to screen for clinically significant mental health problems are those routinely used in primary care. Questionnaires with proven reliability and validity were incorporated where available and otherwise unpublished measures that have been used in previous studies or developed for this study were used. Copies of all unpublished measures can be found in the appendices.

2.4.1 Baseline measures

General Information Questionnaire (GIQ; unpublished)

The 14-item GIQ (Appendix 4) was used to gather demographic information including sex, age, ethnicity, occupational status and native language.

Depression

Patient Health Questionnaire-nine item (PHQ-9; Kroenke et al., 2001; Spitzer, Kroenke, & Williams, 1999)

The PHQ-9 is a widely used and well-validated nine-item self-report measure of depression in the general population. The nine items mirror depressive symptomatology specified by DSM-IV. The PHQ-9 was administered as a screening measure and to match depressive symptomatology across the groups. On the PHQ-9, participants are required to rate how often they have experienced symptoms of depression in the last 7 days on a scale of 0 ('not at all') to 3 ('nearly every day'). Scores range from 0 to 27 with higher scores indicating more severe depression. A cut-off score of 10 or above (indicates moderate depression) is used in primary mental health services (http://www.iapt.nhs.uk) and was used in this study. The PHQ-9 has been shown to be reliable and valid in primary care and in obstetrics-gynecology samples (Kroenke et al., 2001). Criterion validity has been demonstrated by correlating the PHQ-9 with mental health professional interviews indicating 88% sensitivity and 88% specificity for detecting major depression. A strong association with functional status, disability days, and symptom-related difficulty has been illustrated, indicating good construct validity. The PHQ-9 has been shown to have excellent internal reliability (Cronbach's α=.86-0.89) and test-retest reliability (r=.84).

Anxiety

Generalised Anxiety Disorder Assessment – 7 items (GAD-7; Spitzer et al., 2006)

The GAD-7 is a seven-item questionnaire that was used to screen participants for clinically significant levels of general anxiety and to match groups on anxiety symptomatology. The GAD-7 was originally developed as a screening tool for generalised anxiety disorder but has also been shown to screen for other common anxiety disorders including panic disorder, PTSD and social anxiety disorder (Kroenke et al., 2007). The GAD-7 asks participants how often they have been 'bothered' by symptoms of anxiety over the last two weeks, on a scale of 0 ('not at all') to 3 ('nearly every day'). Scores range from 0 to 21 with higher scores indicating greater severity. The GAD-7 is used extensively in primary care as a screening tool for anxiety with a recommended clinical cut-off of 8 (http://www.iapt.nhs.uk) and this was used in this study. The GAD-7 has been shown to have excellent internal consistency (α=.92) and good test-retest reliability (r=.83) (Spitzer et al., 2006). It has been illustrated that as GAD-7 scores increase, functional impairment increases, demonstrating construct validity. The GAD-7 has also been shown to have good convergent validity as it correlates with the Beck Anxiety Inventory (Beck et al., 1988) and the anxiety subscale of the Symptom Checklist-90 (Derogatis et al., 1974), r=.72 and r=.74, respectively (Spitzer et al., 2006).

Spielberger State-Trait Anxiety Inventory-Trait version (STAI-T; Spielberger et al., 1983)

The STAI-T is a 20-item self-report measure of trait anxiety i.e. a tendency to perceive situations as threatening and to increase state anxiety in response to them. The items are statements (e.g. 'I feel nervous and restless') and participants are asked to rate the statements in terms of how they generally feel from 1 ('not at all') to 4 ('almost always'). Higher scores on the scale indicate greater anxiety, with scores ranging from 20 to 80. The STAI-T was administered to ensure that there were no significant differences in trait anxiety between the groups and was investigated as a predictor of intrusion frequency, intrusion distress and PTSD symptoms. The State-Trait Anxiety Inventory is frequently used in research and clinical practice, with internal consistency coefficients ranging from .86 to .95 and test-retest reliability coefficients ranging .65 to .86 (e.g. Spielberger et al., 1983). Studies have also demonstrated construct (Smeets, Merckelbach, & Griez, 1997) and concurrent validity (Spielberger & Reheiser, 2003).

Trauma history and PTSD symptoms

Trauma screener (unpublished)

The trauma screener (Appendix 5) is a self-report checklist of traumatic events. It was administered to match the groups for prior trauma exposure and to identify the participant's most stressful life event for them to reference when completing the baseline IES-R. The trauma screener has been used in previous studies (e.g. Ehlers et al., 1998; White, 2012) and was based on the trauma checklist from the Clinician-Administered Post-traumatic Scale (CAPS; Blake et al., 1990). Participants are asked to answer 'yes' or 'no' to a 22-item checklist of traumatic events (e.g. serious traffic accidents, sexual assault, imprisonment). If they answer 'yes', they are further asked to indicate whether they experienced 'fear, helplessness or horror as a result of the event' and whether they experienced the event as an adult or a child. In addition, participants are asked whether they have experienced a traumatic event not listed and if so what it is. They are then asked to specify which event they would consider to be the most stressful and when this event occurred. In this study, participants were asked to think about this event when completing the baseline IES-R.

Impact of Events Scale-Revised (IES-R; Weiss & Marmar, 1997)

The IES-R is used as a measure of post-traumatic stress symptoms both in research and clinically. Participants were asked to complete the IES-R at baseline as a screening tool and to match the groups for PTSD symptomatology and at one-week follow-up to assess levels of PTSD symptoms in relation to the trauma films. The IES-R is a 22-item measure that asks participants to rate the distress caused by their symptoms from 0 ('not at all') to 4 ('extremely') over the last seven days with respect to the traumatic event that they have experienced. Higher scores indicate greater PTSD symptom severity, with a total score of 88. The trauma screener was used to identify the index trauma for completion of the IES-R at baseline and a clinical cut-off of 33 was used as recommended by Creamer, Bell, & Failla (2003). At follow-up, participants were asked to complete the IES-R with reference to the trauma films. Adapting the IES-R to provide a measure of responses to the trauma films has been done in previous studies (Holmes et al., 2009; Laposa & Alden, 2006; Woud et al., 2012). Laposa & Alden (2006) demonstrated that the Impact of Events Scale correlated with intrusion frequency (r=0.69) further indicating that it is a valid measure for assessing PTSD symptomatology in analogue studies.

The IES-R added a third subscale ('hyper-arousal') to the original Impact of Events Scale (IES; Horowitz, Wilner, & Alvarez, 1979). This was consistent with the addition of a third core symptom cluster in DSM-IV. The IES-R is composed of three subscales: 'intrusion' (items such as "Pictures about it popped into my mind"), 'avoidance' (e.g. "I tried not to talk about it") and 'hyper-arousal' (e.g. "I felt irritable and angry"). The IES-R has been shown to have high internal consistency for the total scale (α=.96) and three subscales (α=.87-.94 for 'intrusion', α=.84-.87 for 'avoidance', and α=.79-.91 for 'hyper-arousal') (Creamer, Bell, & Failla, 2003; Weiss & Marmar, 1997). Test-retest reliability over a six-month period of .87-.94 for the total score (Weiss & Marmar, 1997), .89 for the 'intrusion' scale, .79 for 'avoidance' and .82 for 'hyper-arousal' has been reported (Sundin & Horowitz, 2002). Correlations between the IES-R subscales and the subscales of other measures of PTSD (CAPS and the PTSD Symptom Scale-Self Report; Foa et al., 1993) have also been shown to be higher for the specific subscales (e.g. 'hyper-arousal') on both measures than correlation between subscales (Beck et al., 2008). In terms of discriminative validity, the IES-R has been shown to produce higher scores for individuals with PTSD compared to those without (Beck et al., 2008). It has also been shown that the IES-R correlates highly with measures of PTSD symptomatology (Post-Traumatic Checklist r=.84; Creamer, Bell, & Failla, 2003), anxiety and depression, illustrating concurrent validity (Beck et al., 2008).

2.4.2 Measures during the experiment

Subjective ratings of distress (unpublished)

Visual analogue scales were used to assess subjective distress to the conditioned and unconditioned stimuli (Appendix 6). At the end of the experimental task, participants were asked to rate how distressing they found the stimuli anchored with 0 ('not at all') to 100 ('extremely distressing') at four different time-points in the films. Previous studies have indicated that a general term for emotion (e.g. distress) is a reliable index for gauging shifts in emotion in response to experimental manipulation, and subjective units of distress (SUDS) are commonly used in clinical research and clinical settings (Dalgleish & Yiend, 2006; Richards & Gross, 2000; Schartau, Dalgleish, & Dunn, 2009).

Awareness of contingency

There is debate around whether contingency awareness is necessary to produce the CR (e.g. Dawson, Schell, & Filion, 2007). Some studies have illustrated that awareness of contingency is

necessary to display the conditioned fear response when measured by SCR (Klucken et al., 2009; Tabbert et al., 2006). Participants were therefore asked what they thought the meaning of the CS+ and CS- was, in order to gauge awareness of contingency.

Skin conductance response (SCR)

Skin conductance response (SCR) signals were amplified with a BrainVision Quickamp with 22-bit A/D conversion and a resolution of 71.5 nV (range7150 mV) and digitised at a rate of 125 Hz. SCR was recorded through an auxiliary channel.

Electrodermal activity (EDA) was measured as skin conductance response (SCR) in constant voltage technique using the GSR module produced by Brain Products, which applied a constant voltage of 0.5 V. Sintered silver-silver chloride (Ag-AgCl) cup electrodes were attached to volar surfaces of the medial phalanges of the second and third finger of the non-dominant hand, using MedCaT skin conductance electrode paste (0.05M NaCL saturation) as electrolyte. The electrodes were metal discs set in cylindrical plastic cases that were filled with electrode gel and fixed to the skin using double-sided adhesive circular collars (Boucsein et al., 2012). A bandpass filter of 0.0Hz to 2.00Hz was applied.

EDA data were segmented into 2 second epochs (beginning 2 seconds pre-stimulus) and 8 second epochs (beginning post-stimulus). SCR amplitude was calculated as the difference between the mean SCR level for the 2 seconds preceding stimulus onset and the highest SCR value during the 8 seconds following stimulus onset, as has been done in recent studies (e.g. Pineles, Orr, & Orr, 2009).

2.4.3 Follow-up measures

Intrusions diary (unpublished):

After viewing the films, participants were asked to keep a daily intrusion diary (Appendix 7) for one week to assess the number of intrusive memories and distress experienced. This method of assessing intrusions has been used frequently in analogue trauma paradigms (Holmes & Bourne, 2008). The diary is divided into days and each day is further compartmentalised into periods of time (i.e. morning, afternoon, evening). The diary also contains a reminder of the criteria for an intrusive memory i.e. “a spontaneously occurring memory (an intrusive image or thought which is not deliberately recalled) about any of the scenes that you saw in the films”.

Participants were asked to record the number of intrusions they experienced and their level of subjective distress (rated as 0-10) in relation to each intrusion.

Diary compliance (unpublished)

At follow-up, Participants were asked to rate how accurately and reliably they had completed the intrusions diary to measure diary compliance (see Appendix 8). Participants were asked to rate two questions (accuracy and reliability) on a 10 point likert scale from 0 ('not at all') to 10 ('extremely').

IES-R

Participants were asked to complete the IES-R at follow-up to assess levels of PTSD symptoms in relation to the trauma films. Please see above section for full description.

Response to Memories (R2M) questionnaire (unpublished)

The R2M questionnaire (see Appendix 9) was developed to assess how participants responded to any intrusions that they experienced. The questionnaire has four subscales 'suppression/avoidance', 'dissociation', 'rumination' and 'elaboration' which aim to reflect responses to intrusions which have found to be helpful and unhelpful (Bryant & Harvey, 1995; Clohessy & Ehlers, 1999; Jones & Barlow, 1990; Joseph & Williams, 1997; Laposa & Rector, 2012; Lawrence et al., 1996; Nolen-Hoeksema & Morrow, 1991; Silver, Boon, & Stones, 1983). The first three subscales reflect patterns of responding that are considered to be unhelpful, whilst the last is scored inversely and thought to be a helpful pattern of responding. Higher scores on this scale indicate more dysfunctional responses to the intrusions.

2.5 Materials and tasks

2.5.1 Trauma films

A series of six films were viewed by the participants. The films contained real-life footage of humans and animals in distress. Three of the films depicted scenes of road traffic accidents, one film showed a bull fight, one an elderly gentleman shooting himself and one of a woman being intubated. These trauma films have been used in other studies (Steil, 1997; White, 2012) or were borrowed with permission from Schartau, Dalgleish, & Dunn (2009). Each film contained an introductory voice-over which gave a narrative and context for the film as suggested by previous research (Krans, Naring, & Becker, 2009). Exposure to a traumatic event according to DSM-IV (APA, 1994) includes experiencing or witnessing "events that involved

actual or threatened death or serious injury or a threat to the physical integrity of the self or others" and that the "person's response involved intense fear, hopelessness or horror". Previous studies have illustrated that the films elicit significant levels of distress and horror (White, 2012) and so meet the DSM-IV criteria for a traumatic event. The CS+ was placed in the acquisition films just prior to what was considered to be the worst moment e.g. just before the car crashes. The length of the film clips ranged from 76 to 150 seconds, with a mean length of 108.5 seconds (SD=33.09).

After watching the six trauma films, participants viewed an *intensive presentation phase* in which the image and sound bite from the parts of the films previously paired with the CS+ (the worst moments) was presented again with the CS+. In this phase, the CS- was also paired with neutral images. The CS+ was presented 10 times and the CS- presented 6 times during this phase. The US was displayed for 3 seconds.

In the second stage, the participants viewed either the same films again with an additional narrative (update group), viewed the same set of films again (exposure group) or watched neutral films (neutral group). In the update group, the narrative was expanded to give the participant more information about what happened to the protagonist(s) in the films, aiming to change the meaning of the films and devalue the US. The neutral videos contained scenes of car journeys that were downloaded from the internet.

In order to ensure that the neutral films alone did not provoke significant levels of distress and fear and that the content was rated as neutral, they were viewed by six pilot participants. The participants were asked to rate the neutral films for feelings of distress and fear (measured on a scale of 0 being not at all distressing/fear provoking to 100 being extremely distressing/fear provoking) and rate the valence of the films (with 0 being very positive and 100 being very negative). The average age of these participants was 37.00 (SD 17.27) and there were three males and three females. The films were rated as not producing significant distress (mean 2.50; SD 4.18) or fear (mean 2.50; SD 4.18) and their valence was rated as neutral (mean 48.67; SD 2.16).

2.5.2 Conditioned and unconditioned stimuli

At pre-determined points during the task, the CS+ and CS- appeared in the right hand corner of the screen for two seconds. The CS+ was a red circle (0.5 inches in diameter) and the CS- a blue square (0.7 inches in length).

2.6 Pilot phase

There were two phases of piloting. The first phase was carried out to assess whether the trauma films in the acquisition phase produced the conditioned fear response according to SCR amplitude and subjective ratings. Changes were then made to the protocol and the second piloting phase was completed to further assess whether the stimuli were producing the conditioned fear response and to assess the feasibility of the design. The pilot participants were recruited via email and were all staff or students at King's College London. They all met inclusion criteria for the study and gave written informed consent.

Four participants completed the first phase of piloting. 50% of these participants did not meet minimum response criteria for the conditioned SCR. The mean age of these participants was 27 (SD=1.73) and three of these participants were female and one was male. The *intensive presentation phase* was added to the protocol to attempt to produce a more consistent conditioned response. Nine participants (three per group) completed the second phase of piloting. The mean age of these participants was 27.5 (SD=2.69), eight participants were female and one was male. Eight participants (88%) showed a conditioned SCR above minimum criteria. All participants reported an increase in subjective ratings of distress to the CS+ after viewing the trauma films.

The main aim of the piloting stage was to assess whether the stimuli produced the conditioned fear response at acquisition according to SCR and subjective distress ratings. SCR was inspected visually and 10 out of the 13 pilot participants had produced an SCR amplitude of greater than 0.02μs in response to the unreinforced CS+. Eight out of nine participants in the second phase acquired the conditioned fear response according to this criterion, which was after the addition of the *intensive presentation phase*. For the participants who also viewed the CS- following conditioning (n=6), there was a significant difference between subjective distress ratings to the CS+ (mean=56.67, SD=23.38) and the CS- (mean=0, SD=0) following

conditioning, *T*=-2.21, p=.027, r=-.64. These results indicated that the acquisition phase successfully evoked the differential conditioned fear response.

The pilot participants were also compared in terms of their baseline characteristics and outcome measures for each group. The mean age of participants in the update group was 28.14 (SD=2.68), in the exposure group was 26.67 (SD=1.15) and in the neutral group was 26.33 (SD=2.52). Exploratory analyses indicated that there were no significant differences between the groups in the baseline characteristics (age, sex, IES-R, GAD-7, PHQ-9, STAI-T, trauma screener) or outcome measures (intrusion frequency, intrusion distress, IES-R at follow-up, R2M) (see Appendix 10).

2.7 Procedure

Please see Figure 4 for an overview of the study procedure. Participants were recruited using an email circular advertising the study (Appendix 1) and inviting them to contact the researcher if they were interested in participating. The email circular provided some practical information about the study and a rationale. People who expressed an interest in completing the study were sent the information sheet (Appendix 11) and given an opportunity to ask any questions prior to completing the screening questionnaires. At this stage, they were asked whether they had completed similar trauma studies and whether they mostly work in A&E or hospitals; if they answered yes to either of these questions then they were thanked for their interest but excluded from the study. Participants were reminded that they could withdraw from the study at any time without giving a reason.

After having time to read and consider the information sheet, participants were asked to complete the baseline measures and screening questionnaires (GIQ, STAI-T, PHQ-9, GAD-7, trauma screener and IES-R) on-line before attending the session. Those who were excluded from the study due to clinically significant levels of anxiety, depression or current PTSD symptoms, were informed that they cannot complete the study due to their scores on these questionnaires. They were offered support by the researcher (a clinical psychologist in training with experience of working with people with anxiety, depression and PTSD) and, if they were interested, signposted to relevant agencies to receive help.

Participants who met inclusion criteria were invited to attend a one hour session to complete the experimental task at the Institute of Psychiatry, Kings College London. Participants were randomly allocated to one of the three groups before attending the session. The experimental session took place in a temperature-controlled quiet room in the Institute of Psychiatry. The participant, equipment and the researcher were located in the same room. The room was arranged to seat the participant in front of the researcher and recording equipment, so that the participant could not see either during the conditioning procedure.

Participants were given another opportunity to read the information sheet, ask any questions and then informed written consent (Appendix 11) was obtained before testing began. The SCR equipment was then attached to the participant for the duration of the session and their responses checked before beginning. Participants were seated in a comfortable chair approximately 20 inches away from a 15 inch computer screen in the corner of the testing room. Participants were asked to make themselves as comfortable as possible before testing began. All participants were given the following instructions:

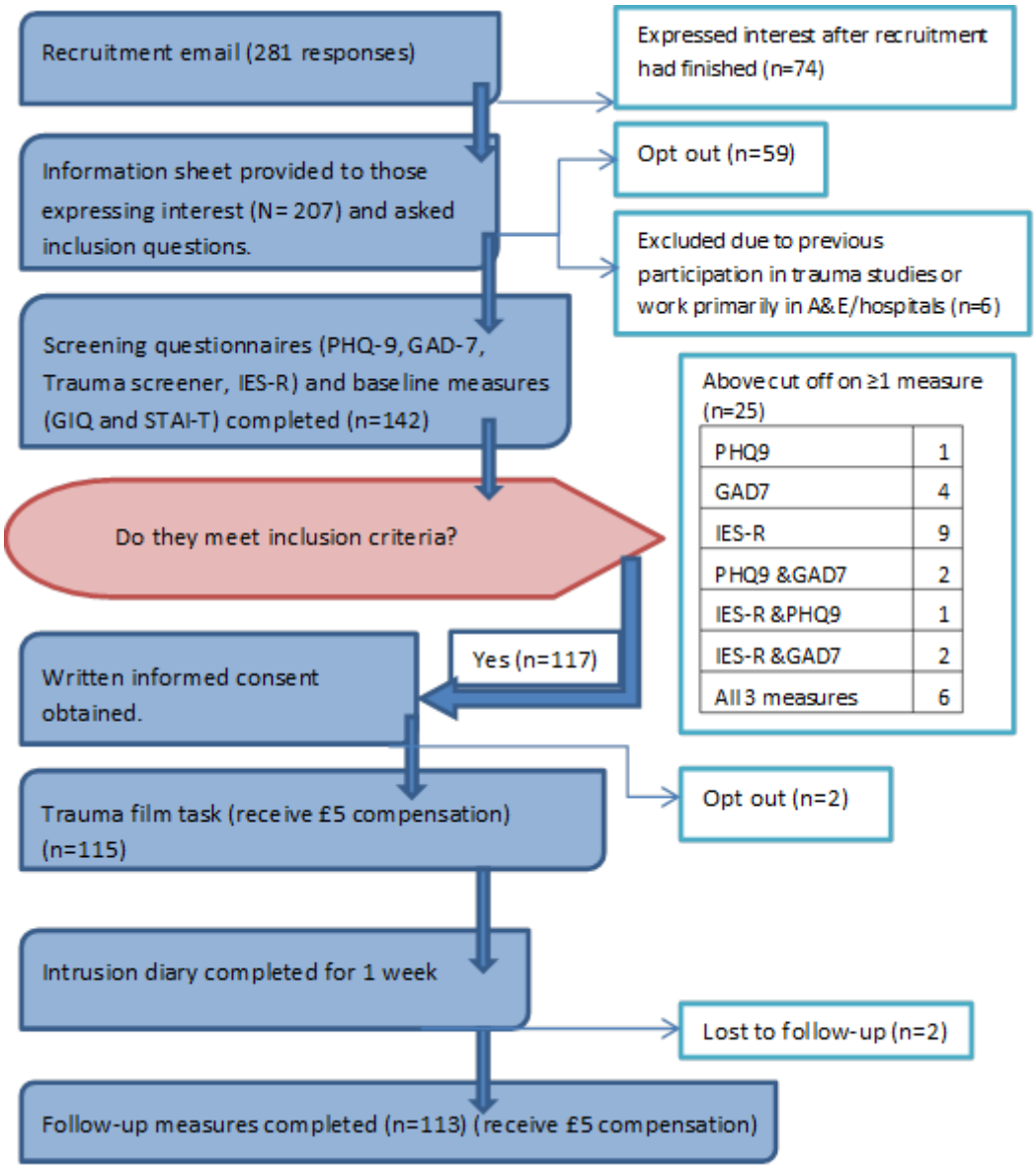
"You will be watching a series of film clips that last approximately 40 minutes. As much as possible, do not move or talk during this time. You may find that your concentration starts to fade and so, as much as possible, try to keep engaged with the films. This hand (point to their non-dominant hand with the SCR sensors attached) is very important and so try not to move this hand at all. There may be times during the films when you think that the films have finished but keep watching the screen and I will let you know when the task is complete. I would like you to try and work out the meaning of the red dot and the blue square contained in the films and I will be asking you some questions about the films once they have finished. Have you got any questions?"

The participants were given an opportunity to ask any final questions and when they had confirmed that they were comfortable and their physiological response had settled, the films began. Once they had completed the experimental task, participants were asked for their subjective ratings of distress to the CS+ and CS- and what they thought the meaning of the CS+ and CS- was. Participants were requested to keep an intrusion diary for one week. They were asked to keep a note throughout the day when they experienced an intrusion and to specify a time each day when they would complete the diary. At this point, the difference between spontaneously occurring memories (intrusions) and consciously recalled memories of the films

was discussed and participants were asked only to record the former. Participants were also asked whether they thought that they would have any difficulties completing the diary and any obstacles were problem-solved with the researcher before leaving the session. After completing the session, participants were paid £5 as compensation for their time.

One week after completing the experimental task, participants were sent an email to thank them for completing the study and to ask them to complete the follow-up questionnaires on-line. Participants were given the choice of submitting the diary electronically, by post or meeting the researcher. After returning the diary and completing the follow-up questionnaires, participants were given a further £5 as compensation for their time. All participants were asked to complete the follow-up measures and were paid the full £10 whether or not they demonstrated a conditioned SCR above minimum response criteria.

Figure 4: Procedure diagram



2.8 Analogue experimental task

Figure 5 summarises the experimental design. There were a relatively small number of acquisition and US devaluation trials in this experiment. The rationale for this was to try to prevent habituation during acquisition so that subjects would hopefully still show electrodermal responsivity during the test phase (Lovibond, Davis & O'Flaherty, 2000). The participants were first shown a single presentation of the to-be-conditioned stimulus CS+ without the US. All participants then took part in phase 1: *acquisition.*

Acquisition: participants watched a series of six short film clips with introductory narratives and the CSs embedded within them. The CS+ was inserted into the films to signal the most distressing part of the film. So, for example, in a RTA clip, the CS+ appears on the screen just prior to the crash occurring. After viewing the films the *intensive presentation phase* began in which the CS+ was paired with the single image and noise from the part of the film with which it was previously paired. The CS- was paired with neutral images, for example an image of a tree. In this phase there were two unreinforced presentations of the CS+ (mean denoted as CS+ACQ) and one unreinforced presentation of the CS- (CS-ACQ).

US devaluation*:* reinforced CSs were not presented in this phase. The participants were randomly allocated to one of three US devaluation groups:

1. Update: participants viewed the same films again but the introductory passage was elaborated to contain additional information about what happened to the protagonists in the films. For example, a film clip and introductory passage depicting a young woman who is six months pregnant and is being intubated following a car crash where her friend is driving is updated with information that *"the friend survives with injuries, the pregnant woman has to be treated at the accident scene by an emergency doctor. She is airlifted to hospital to undergo surgery. She is in hospital for several weeks after the accident to minimise her movement and to monitor her pregnancy. She and her baby survive and three months after the accident she gives birth to a happy healthy baby girl."*
2. Exposure: the trauma films with the introductory passages were viewed again by participants.
3. Neutral: participants viewed non-traumatic films of related content, for example cars driving on a motorway.

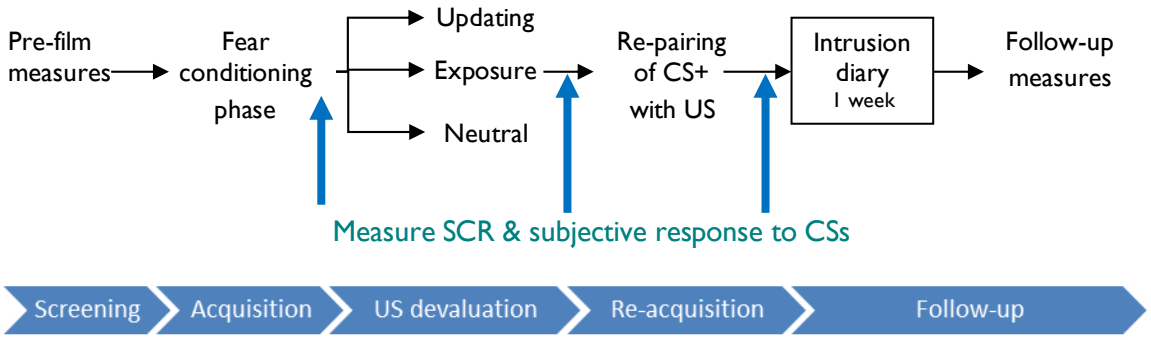
The participants were then presented with a single unreinforced CS+ (CS+USDe).

Re-acquisition: the CS+ and CS- are re-paired with their respective US and noUS by presenting the *intensive presentation phase* again. Two unreinforced CS+s (mean denoted as CS+REACQ) and one unreinforced CS- (CS-REACQ) followed this phase.

After the films are finished, the participants were asked to rate the level of distress they experienced to the unreinforced CS+ and CS- following the acquisition, US devaluation and re-acquisition phases and were asked to detail what they thought the meanings of the CS+ and CS- were.

Follow-up: At the end of the session, the intrusion diary was discussed with participants and they were provided with a paper diary to take home and offered to be emailed an electronic version if that was preferable to them. They were asked to complete the diary every day over the following week, to record any spontaneously occurring memories that they experienced related to the film clips. Participants were asked to fill in the follow-up questionnaires on-line via a link provided one week later by the researcher. Participants were asked to return the diary and complete the follow-up questionnaires in order to receive final payment.

Figure 5: Overview of experimental procedure



2.9 Data analyses

2.9.1 Skin conductance response (SCR)

Traditionally, studies separated SCR into two components, first (FIR) and second (SIR) interval responses. However, recent studies do not support this convention and recommend that SCR scores are taken from data obtained across the entire CS/UCS interval (Pineles, Orr, & Orr, 2009). Pineles, Orr, & Orr (2009) highlight several advantages of using the entire CS/UCS interval. These include making no assumptions about where the response is likely to occur within the interval and so ensuring that the CR is not underestimated e.g. if the response occurs at the FIR/SIR boundary or shifts over trials. It is also a simpler way to score the data and overcomes the conceptual dilemma of where to place an arbitrary boundary e.g. two six-second intervals for a 12-second CS/US interval compared to two four-second intervals for an eight-second CS/US interval. This approach has been employed by similar studies investigating

human psychophysiology (Kindt & Soeter, 2011; Milad et al., 2005; Orr et al., 2000; Pineles, Orr, & Orr, 2009)

A minimum response criterion of 0.02μs was applied in this study, as is indicated by similar studies (Kindt & Soeter, 2011; Schiller et al., 2010; Schiller et al., 2008; Soeter & Kindt, 2011). In terms of the participants who do not show an SCR above this minimum on the acquisition trials, the literature indicates that options for analysis include excluding these participants from analysis (e.g. Schiller et al., 2010) or scoring these entries as zero and including them in the analysis (e.g. Kindt & Soeter, 2011; Pineles, Orr, & Orr, 2009). The data were analysed using both methods.

2.9.2 Statistical analysis

Data were analysed using SPSS for Windows version 19. A two-tailed significant level of α=0.05 was used throughout analysis. The data were assessed for normality and homogeneity of variance. Normality was assessed by calculating z-scores for skewness and kurtosis and using a recommended cut-off of 2.58 (Field, 2009). Variables that violated parametric assumptions were transformed (log 10, square root or reciprocal) and their normality re-assessed. Reported values are all untransformed in order to be more readily readable. Non-parametric tests were used to analyse variables that broke assumptions of normality following transformation. Levene's test was used to assess homogeneity of variance.

Differences between the groups in terms of baseline characteristics were analysed using Chi-squared analysis for categorical variables and a combination of one-way ANOVAs and their non-parametric equivalent for age, screening questionnaires, CS+ACQ (SCR and distress ratings) and SCR during the films and diary compliance. To compare the changes in SCR amplitudes and distress ratings across the experimental paradigm, a mixed-measures ANOVA would have ideally been conducted. However, the data were heavily skewed and violated all assumptions even after transformation. Therefore, Kruskal-Wallis tests were used that compared the difference scores between the phases for the SCR amplitudes and distress ratings for each group. A multivariate analysis of variance (MANOVA) was used to compare intrusion frequency, intrusion distress and IES-R scores for the three groups. A multivariate analysis of covariance (MANCOVA) was used to analyse the relationship between an individual's conditioned acquisition response, trait anxiety and maladaptive responses to

memories of the trauma films and their intrusion frequency, intrusion distress and PTSD symptomatology.

Effect sizes were calculated by using Pearson's r or partial eta-squared (partial η^2). For Pearson's r, 0.10 indicates a small effect, 0.30 a medium effect and 0.50 a large effect (Field, 2009). For partial eta-squared, values of 0.01, 0.06 and 0.14 indicate small, medium and large effects respectively (Cohen, 1988).

3. Results

3.1 Group comparisons at baseline

The groups were compared in terms of demographic factors, self-reported anxiety, depression and trauma history and conditioned acquisition responses and SCR during the acquisition films. The groups were also compared in terms of their self-reported diary compliance at follow-up. There were 37 participants in the update group, 41 in the exposure group and 37 in the neutral group.

Demographics

The mean age of participants was 25.49 (SD 8.34) in the update group, 27.12 (SD 8.86) in the exposure group and 26.97 (SD 7.51) in the neutral group. Kruskal-Wallis analysis showed no significant difference in age between the three groups $H(2)=2.01$, p=.90. In all three groups, most participants were female: there were 27 females in the update group (73.0%), 32 (78.0%) in the exposure group and 28 (75.7%) in the neutral group. There was no significant difference in sex between the three groups as indicated by Chi-squared analysis: $\chi^2(2)=0.27$, p =.96.

Anxiety, depression and history of trauma

There were no significant differences between the groups on any of the baseline measures: trait anxiety (STAI-T), depression (PHQ-9), general anxiety (GAD-7), PTSD symptoms (IES-R), and previous trauma exposure (trauma screener) (see Table 1).

Table 1: Baseline measures

	Update Mean (SD) (n=37)	Exposure Mean (SD) (n=41)	Neutral Mean (SD) (n=37)	1-way ANOVA
STAI-T	34.54 (8.17)	35.78 (9.44)	33.76 (9.11)	$F(2,112)=0.51$, p=.61
IES-R	6.81 (8.68)	7.48 (8.69)	6.14 (8.27)	$F(2,112)=0.21$, p=.81
PHQ-9	1.49 (1.98)	2.00 (2.33)	1.65 (2.37)	$F(2,112)=0.99$, p=.37
GAD-7	1.57 (1.74)	1.70 (1.97)	1.35 (1.98)	$F(2,112)=0.85$, p=.43
Trauma screener	2.23 (1.87)	1.83 (1.75)	2.24 (2.08)	$F(2,112)=0.39$, p=.68

Data were log transformed prior to analysis. Untransformed values are reported.

SCR amplitude and subjective distress ratings to CS+ following acquisition

All participants viewed the same set of acquisition films and so there should be no differences between the groups following acquisition. There was no significant difference between the groups in terms of ratings of distress or SCR amplitude to the CS+ACQ (please see Table 2 for

means and standard deviations [SD], medians and interquartile ranges [IQR] and statistical tests). In addition, participants' SCR amplitude during the worst parts of the acquisition films did not differ significantly between the groups (see Table 3). The difference between the SCR amplitudes for film 4 approached significance, $H(2)=5.53$, p=.063. However, this was also the film that produced the smallest mean SCR amplitude and so is unlikely to significantly affect SCR to the CSs at test and group differences for the other five films did not approach significance.

Table 2: Subjective distress ratings and SCR following acquisition

CS+ACQ	Update (n=37)	Exposure (n=41)	Neutral (n=37)	Comparison between groups
Distress rating CS+ Mean (SD)	53.02 (31.72)	41.83 (27.29)	42.97 (25.94)	One way ANOVA $F(2,112)=1.78$, p=.17
SCR Median (IQR)	0.056 (0.23)	0.13 (0.38)	0.051 (0.03)	Kruskal-Wallis $H(2)=1.49$, p=.48

Table 3: SCR during acquisition films

	Update (n=37) Median (IQR)	Exposure (n=41) Median (IQR)	Neutral (n=37) Median (IQR)	Kruskal-Wallis
Film 1	0.34 (0.47)	0.31 (0.55)	0.25 (0.53)	$H(2)=1.70$, p=.43
Film 2	0.16 (0.40)	0.22 (0.53)	0.11 (0.49)	$H(2)=0.56$, p=.76
Film 3	0.36 (0.71)	0.33 (0.88)	0.25 (0.89)	$H(2)=0.11$, p=.95
Film 4	0.10 (0.15)	0.084 (0.38)	0.008 (0.05)	$H(2)=5.53$, p=.063
Film 5	0.21 (0.38)	0.21(0.51)	0.11 (0.54)	$H(2)=0.26$, p=.88
Film 6	0.07 (0.37)	0.09 (0.51)	0.08 (0.29)	$H(2)=0.31$, p=.86

Follow-up measures: diary compliance

The groups did not differ significantly in their self-reported accuracy and reliability in completing the intrusion diary over the week following the experiment (see Table 4).

Table 4: Self-reported diary compliance at follow-up

Diary Compliance	Update (n=37) Mean (SD)	Exposure (n=41) Mean (SD)	Neutral (n=37) Mean (SD)	ANOVA
Accurate	8.68 (1.20)	8.29 (1.23)	7.86 (1.87)	$F(2,111)=1.92$, p=.15
Reliable	2.3 (1.94)	2.12 (1.58)	2.17 (1.65)	$F(2,111)=0.72$, p=.93

Data were log transformed. Untransformed values are reported.

Baseline characteristics: summary

There were no significant differences between the groups on any of the baseline measures. Participants did not differ in terms of demographic variables, self-reported anxiety, depression, trauma history and PTSD symptoms before beginning the experiment. Participants all received

the same acquisition conditioning and there were no significant differences between the groups in SCR or self-reported distress to the CS+ACQ or SCR during the acquisition films. The groups also did not differ in terms of their reliability and accuracy in completing the intrusions diary. This suggests that the groups were well-matched on criteria related to the hypotheses examined in this study.

3.2 Analysis of hypotheses

3.2.1 Fear conditioning using trauma film stimuli

Hypothesis 1 (H1): Participants will produce the conditioned fear response when trauma film stimuli are used as the US. Conditioning is defined as having an SCR amplitude to the unreinforced CS+ that is greater than 0.02µs and that SCR and subjective distress ratings are greater to the CS+ than the CS-.

The mean conditioned response for the two unreinforced CS+s in the acquisition phase was calculated (CS+ACQ). 75 of the 115 cases (65%) produced a mean amplitude change greater than 0.02µs. SCR to the CS+ACQ was significantly different to SCR to the CS-ACQ, *T*=294, p=.00 r=-.49. There was also a significant difference between subjective distress ratings to the CS+ACQ compared to the CS-ACQ, *T*=13, p=.00, r=-.57, across all cases (n=115). Please see Table 5 for results. The paradigm was also successful in eliciting intrusive memories of the films with 107 of 113 (95%) participants reporting at least one intrusive memory of the films.

Table 5: CS+ACQ compared to the CS-ACQ using SCR and subjective distress ratings

	CS+ACQ Median (IQR)	CS-ACQ Median (IQR)	Wilcoxon signed-rank test
SCR	0.20 (0.41)	0.09 (0.06)	*T*=294, p=.00, r=-.49
Subjective rating (all cases)	50.00 (50.00)	0 (0.00)	*T*=13, p=.00, r=-.57

H1: summary

Fear conditioning, as measured by SCR amplitude and subjective distress ratings, can occur using trauma film stimuli as the US. Approximately two thirds of participants showed a SCR to the CS+ACQ above minimum criteria and this was significantly different to the response to the CS-ACQ (medium to large effect, r=.49). There was also a significant difference in subjective distress ratings (large effect, r=.57) to the CS+ACQ and CS-ACQ. The majority of participants (95%) also reported at least one intrusive memory of the film.

3.2.2 Effect of experimental manipulation on the conditioned fear response

Hypothesis 2 (H2): (A) Participants in the update group will have the largest reduction in physiological response and subjective distress ratings following the US devaluation phase compared to those in the exposure and neutral groups. (B) Participants in the update group will have the smallest increase in conditioned response and distress ratings following the re-acquisition phase.

A mixed ANOVA with stage (CS+ACQ, CS+USDe, CS+REACQ) as the within-subjects factor and group as the between-subjects factor would have ideally been conducted. However, the data were heavily skewed and violated assumptions even after transformation. Therefore, difference scores were calculated and analysed using non-parametric tests.

Results were first analysed by excluding participants who did not meet the minimum response criteria of 0.02μs at acquisition (e.g. Schiller et al., 2010) and then all cases were analysed with those cases not meeting minimum criteria being scored as zero and retained in the analysis (e.g. Kindt & Soeter, 2011; Pineles, Orr, & Orr, 2009) (see Table 7 & 8 and Figure 6). When cases that did not meet minimum response criteria were excluded, there were still no significant differences between the groups on the baseline measures (see Appendix 12).

US devaluation

The difference between the SCR amplitude and subjective distress ratings to the CS+ following US devaluation (CS+USDe) and following acquisition (CS+ACQ) was calculated (CS+USDe minus CS+ACQ).

Only cases meeting minimum response criteria at acquisition

When cases that did not produce an SCR amplitude to the CS+ACQ of greater than 0.02μs were excluded from analysis, there were 23 cases in the update group, 29 in the exposure group and 23 in the neutral group. Analysis, using the Kruskal-Wallis test, revealed that there was a significant difference, $H(2)=9.66$, $p=.008$, between the groups. Mann-Whitney tests were used to follow up this finding and indicated a significant difference between the update and exposure groups ($U=164$, $p=.002$, $r=-.43$) and no significant differences between the other groups. Surprisingly, the difference between the SCR to the CS+USDe and CS+ACQ was not in the expected direction for the update group. In the update group, SCR on average increased from CS+ACQ to CS+USDe where it was expected to decrease. The exposure group and neutral groups showed a fall in SCR from CS+ACQ to CS+USDe as predicted.

All cases included in analysis

Analysis revealed similar findings when all the cases were included in the analysis. Kruskal-Wallis tests revealed a significant difference between the three groups, $H(2)=8.78$, p=.012, and Mann-Whitney tests were used to follow up this finding. There was a significant difference between the updating and exposure groups, $U=446.5$, p=.003, r=-.33, with no significant difference between the other groups. The difference between the exposure and neutral groups was at trend level significance $U=927.5$, p=.091. The difference between the CS+USDe and CS+ACQ in the update group was again not in the expected direction with, on average, participants having larger SCR amplitudes to the CS+USDe than the CS+ACQ. The neutral and exposure groups' difference scores were in the expected direction.

Subjective distress

Analysis, using Kruskal-Wallis, indicated a significant difference between the groups in changes in subjective distress scores between CS+USDe and CS+ACQ, $H(2)=10.29$, p=.006, and this was in the expected direction. Mann-Whitney tests were used to follow up this finding. There was a significant difference between the updating and exposure groups, $U=1069$, p=0.002, r=-.36, and between the updating and neutral groups, $U=482$, p=.027, r=-.26,. There was no significant difference between the exposure and neutral groups. Baseline analysis illustrated that there was no significant difference between the groups in subjective distress ratings to the CS+ACQ.

Therefore, as predicted, there was a greater reduction in subjective ratings of distress from CS+ACQ to CS+USDe in the update group compared to the other two groups. This is in the opposite direction to the changes in SCR. This implies that the update devaluation was the most effective in reducing subjective ratings of distress.

SCR amplitude during US devaluation films

Participants' SCR during each US devaluation film were compared for the update and exposure groups. The neutral group was not included in this analysis as they viewed neutral films, whereas the exposure and update group viewed the same films. There were no significant differences between the update and exposure groups in SCR amplitude during the US devaluation films (see Table 6). There was a trend level significant difference in film 9, $U=942$, p=0.066, with the exposure group having a larger SCR than the update group. Overall, this suggests that updating the meaning of the films had no immediate impact on the physiological fear response when viewing the films.

Table 6: SCR amplitudes during US devaluation films

	Update (n=37) Median (IQR)	**Exposure (n=41) Median (IQR)**	**Mann-Whitney**
Film 7	0.051 (0.29)	0.064 (0.33)	*U*=850, p=.36
Film 8	0.14 (0.7)	0.092 (0.4)	*U*=755, p=.98
Film 9	0.084 (0.3)	0.18 (0.67)	*U*=942, p=.066
Film 10	0.006 (0.06)	0.006 (0.05)	*U*=717, p=.67
Film 11	0.037 (0.13)	0.017 (0.21)	*U*=754, p=.97
Film 12	0.067 (0.37)	0.029 (0.24)	*U*=631, p=.20
Overall	0.14 (0.26)	0.17 (0.29)	*U*=811, p=.60

Re-acquisition

Re-acquisition was calculated by taking the difference between the mean SCR to the unreinforced CS+ presentations after the re-acquisition phase (CS+REACQ) and the US devaluation phase (CS+USDe).

SCR

There was no significant difference between the groups from US devaluation to re-acquisition in their conditioned SCR for only those cases meeting minimum response criteria at acquisition, $H(2)=4.74$, p=.093, or for all cases, $H(2)=3.71$, p=.16. This is not surprising due to the lack of reduction in the fear response observed in the neutral and update groups. Indeed all groups showed a mean decrease in SCR to the CS+REACQ from the CS+USDe.

Subjective distress

The groups did not differ significantly on changes in subjective distress ratings from US devaluation to re-acquisition, $H(2)=0.72$, p=.70. However, in line with predictions, participants reported an increase in distress to the CS+REACQ from the CS+USDe. At the end of the experimental phase, there was no significant difference in overall ratings of distress between the three groups, $F(2, 112)=0.01$, p=.991.

Table 7: Changes in SCR (only cases that meet minimum criteria) and distress ratings for the CS+ for each group over the three phases

	Measure	CS+ACQ	CS+USDe	CS+REACQ
Update median (IQR)	SCR>0.02 (n=23)	0.20 (0.31)	0.25 (0.85)	0.11 (0.17)
	Distress ratings (n=37)	60.00 (52.50)	40.00 (35.00)	50.00 (43.50)
Exposure median (IQR)	SCR>0.02 (n=29)	0.22 (0.44)	0.051 (0.24)	0.03 (0.19)
	Distress ratings (n=41)	40.00 (40.00)	40.00 (57.50)	40.00 (52.50)
Neutral median (IQR)	SCR>0.02 (n=23)	0.15 (0.42)	0.052 (0.4)	0.05 (0.23)
	Distress ratings (n=37)	50.00 (45.00)	30.00 (100.00)	50.00 (50.00)

Figure 6: Changes in SCR (only cases that meet minimum criteria) and distress ratings for the CS+ for each group over the three phases

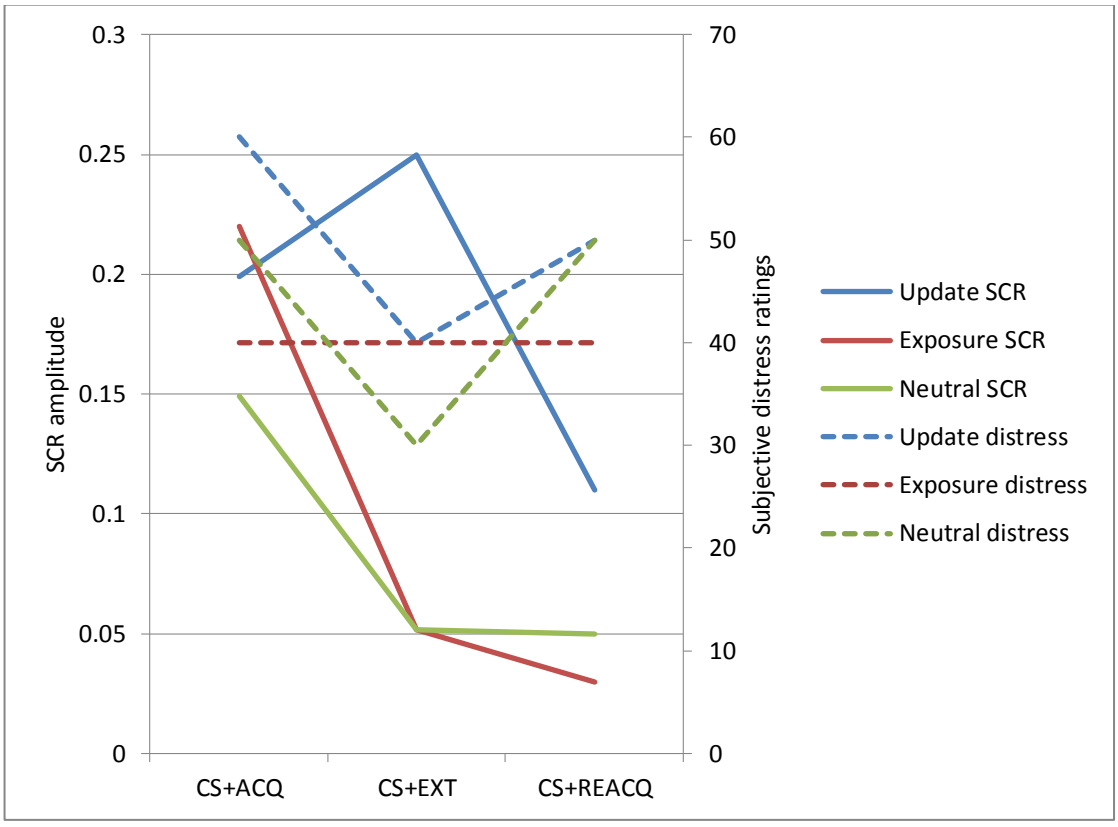


Table 8: Changes in SCR (all cases) across the three phases for each group

All cases	CS+ACQ	CS+USDe	CS+REACQ
Update median (IQR) (n=37)	0.056 (0.23)	0.15 (0.58)	0.08 (0.16)
Exposure median (IQR) (n=41)	0.13 (0.38)	0.039 (0.17)	0.03 (0.15)
Neutral median (IQR) (n=37)	0.051 (0.03)	0.026 (0.24)	0.03 (0.14)

Awareness of contingency

Whether contingency awareness is necessary for fear conditioning is debated (Dawson, Schell, & Filion, 2007). A small number of participants (n=11) did not report the correct awareness of the CS+/US contingency. Excluding these participants from analysis did not alter the results and so they were retained in the analysis.

H2: Summary

The exposure group had a significantly larger reduction in SCR amplitude to the unreinforced CS+ following US devaluation than the update group (medium effect, r=0.41). Interestingly, participants in the update group showed an increase in SCR to the CS+ from acquisition to US devaluation. However, the update group had a significantly larger drop in subjective ratings of distress to the CS+ following US devaluation than both the exposure (medium effect, r=-.36) and neutral groups (small to medium effect, r=-.26). This implies that updating the meaning of the US reduces subjective distress but increases SCR to the CS+. There were no significant differences following re-acquisition, in terms of SCR or subjective distress ratings to the CS+, between the groups. This is not surprising given the lack of reduction in the conditioned fear response.

3.2.3 Effect of experimental manipulation on intrusion frequency, intrusion distress and PTSD symptoms

Hypothesis 3 (H3): Participants in the update group will experience fewer intrusions, be less distressed by them and have fewer PTSD symptoms in the week following the trauma paradigm than the exposure or neutral groups.

A MANOVA was conducted with intrusion frequency, intrusion distress and IES-R follow-up score as dependent variables and group as a fixed factor (see Table 9 and Figure 7). A MANOVA was used to protect against multiple testing and because it is expected that the outcome measures are similar. This revealed, using Pillai's trace, a significant effect of group on the dependent variables, V=0.18, $F(6,218)=3.50$, p=.002, partial $\eta^2=.088$. Separate univariate ANOVAs were conducted on the dependent variables and found significant differences between the groups on intrusion frequency and IES-R scores. Independent t-tests were used to examine individual group differences.

There was a significant difference in intrusion frequency between the groups, $F(2, 110)=5.99$, p=.003, r=.31. Follow-up analysis using independent t-tests revealed that the update group

reported significantly fewer intrusions than either the exposure group, *t*(75)=-3.35, p=.001, r=.36, or the neutral group, *t*(71)=-2.61, p=.011, r=.30. There was no significant difference in intrusion frequency between the exposure and neutral groups, *t*(74)=.51, p=.61. The difference in intrusion distress between the groups approached significance, *F*(2, 110)=2.67, p=.074; inspection of the means suggests that the update group reported less intrusion distress than the neutral and exposure groups.

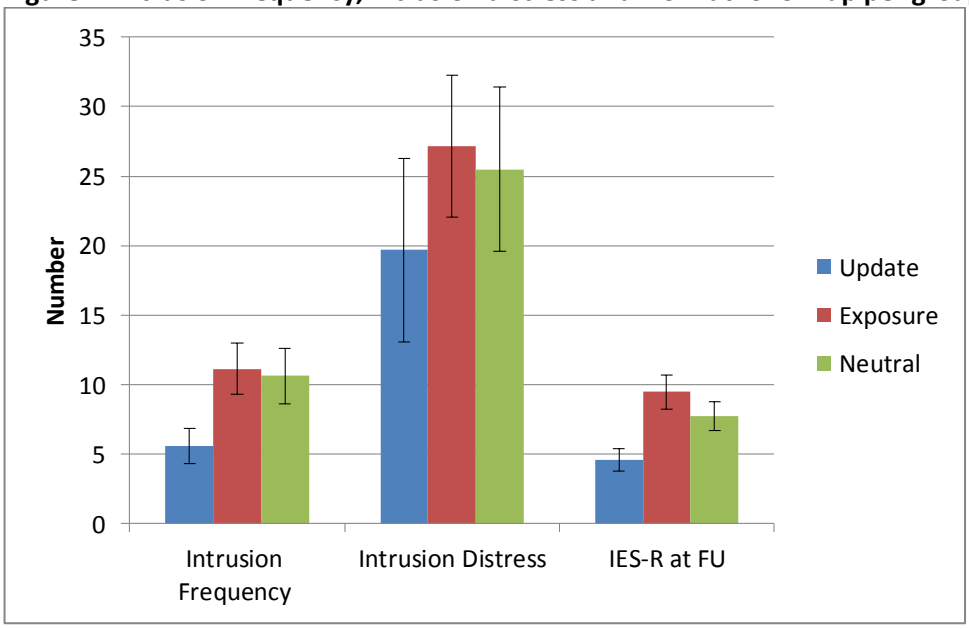
The groups differed significantly in terms of IES-R scores at follow-up, *F*(2, 110)=7.70, p=.001, r=.35. Independent t-tests revealed that the update group had significantly lower IES-R follow-up scores than either the exposure group, *t*(75)=-3.73, p=.00, r=.40, or the neutral group, *t*(71)=-2.71, p=.008, r=.31. There was no significant difference in IES-R scores between the exposure and neutral groups, *t*(74)=1.01, p=.32.

Table 9: Intrusion frequency, intrusion distress and IES-R for each group.

	Update mean (SD) (n=37)	**Exposure mean (SD) (n=40)**	**Neutral mean (SD) (n=36)**	**1-way ANOVA (2 tailed)**
Intrusion frequency	5.60 (7.66)	11.15 (11.45)	10.64 (11.86)	*F*(2, 110)=5.99, p=.003, r=.31
Intrusion distress	19.68 (40.25)	27.15 (32.18)	25.50 (35.63)	*F*(2, 110)=2.67, p=.074
IES-R at FU	4.54 (4.94)	9.47(7.73)	7.72 (6.28)	*F*(2, 110)=7.70, p=.001, r=.35

Intrusion frequency, distress and IES-R scores were log transformed. Untransformed values are reported.

Figure 7: Intrusion frequency, intrusion distress and IES-R at follow-up per group



Means and standard errors are shown.

H3: summary

There were significant differences between groups in terms of intrusion frequency in the week following the trauma film paradigm and scores on the IES-R at follow-up. The update group experienced significantly fewer intrusions and lower PTSD symptomatology than the exposure (intrusion frequency: medium effect, r=.36; IES-R: medium effect, r=.40) and neutral groups (intrusion frequency: medium effect, r=.30; IES-R: medium effect, r=.31). The difference in intrusion distress between the groups was at trend level significance. These results imply that verbally devaluing the trauma films with further meaning reduces intrusion frequency and PTSD symptomatology compared to further exposure to the films or watching neutral films.

3.2.4 Predictors of intrusion frequency, intrusion distress and PTSD symptoms

Hypothesis 4 (H4): Participants who have a smaller conditioned fear response following acquisition will experience fewer intrusions, be less distressed by them and report fewer PTSD symptoms in the week following the trauma paradigm.

The differential conditioned fear response following acquisition (CSdiffACQ) was calculated as the mean SCR amplitude to the unreinforced CS+s (CS+ACQ) minus the SCR amplitude to the unreinforced CS- (CS-ACQ). This differential SCR was used in order to control for individual differences in SCR level. An MANCOVA was conducted with group as a categorical variable, intrusion frequency, intrusion distress and IES-R as the dependent variables and CSdiffACQ as a covariate. The assumptions were met including the residuals being normally distributed, independence of treatment effect and covariate (as at the acquisition stage all groups had viewed identical films) and the assumption of homogeneity of regression slopes (as there was no significant interaction effect between group and CSdiffACQ).

Analysis (n=113), using Pillai's trace, revealed a significant effect of the covariate, CSdiffACQ, on the dependent variables, V=0.12, $F(3,107)=3.48$, p=.004, partial $\eta^2=.12$. There was a significant effect of group after controlling for CSdiffACQ, V=0.18, $F(6,216)=3.46$, p=.002, partial $\eta^2=.090$. The analysis indicated that there was a positive relationship between CSdiffACQ and the dependent variables (see Figure 8).

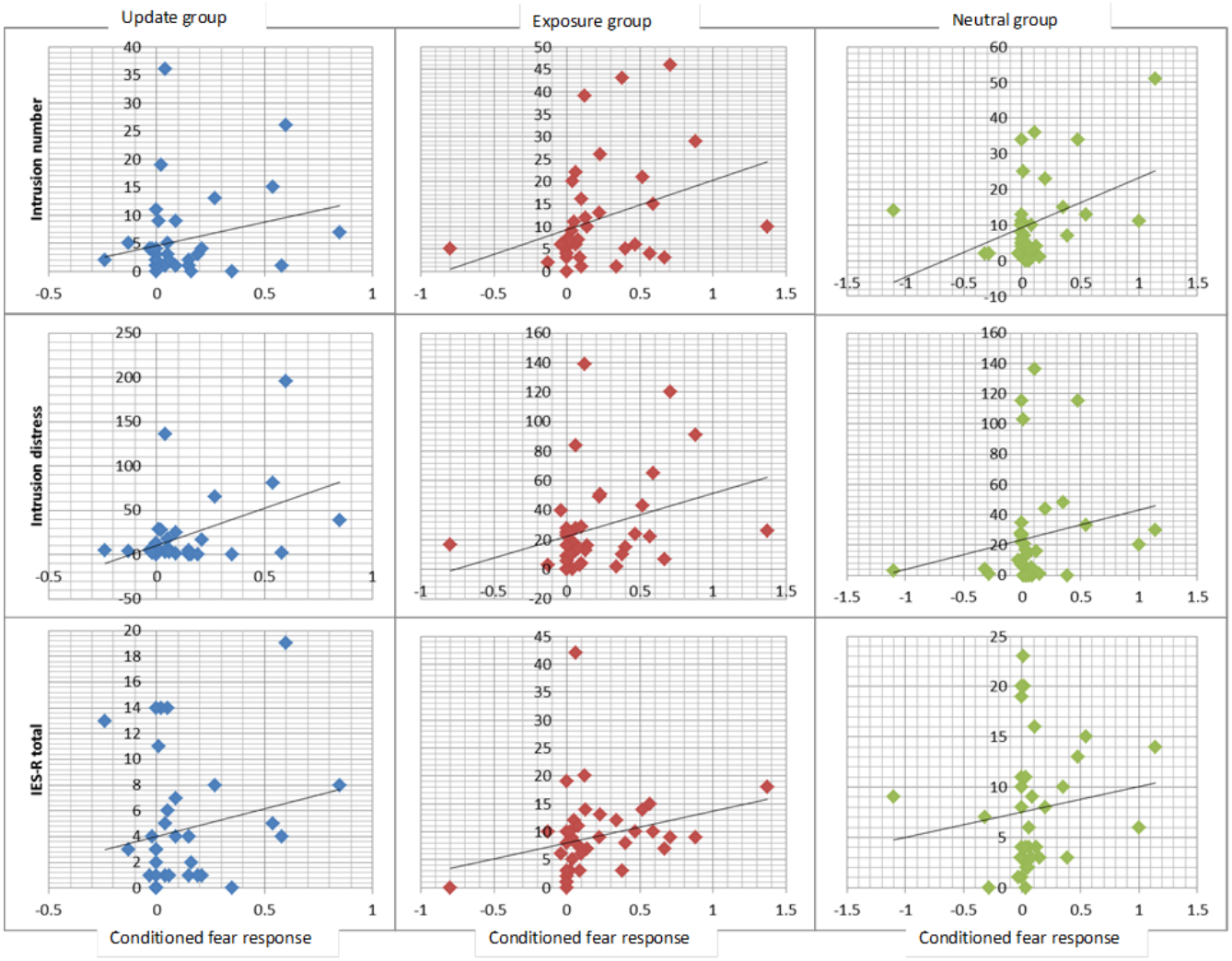
Follow-up tests revealed that CSdiffACQ was significantly related to intrusion frequency [$F(1, 109)=1.49$, p=.002, partial $\eta^2=.085$] intrusion distress [$F(1, 109)=3.52$, p=.002, partial $\eta^2=.084$] and IES-R score [$F(1, 109)=1.15$, p=.004, partial $\eta^2=.075$]. This implies that participants who

acquired a stronger conditioned fear response (i.e. showed larger amplitude SCR to the CS+ following acquisition) experienced more intrusions, were more distressed by them and experienced more PTSD symptoms following the trauma film paradigm. This finding is perhaps in contrast to the earlier findings (H2 and H3) indicating that the update group had both the fewest intrusions/PTSD symptoms and higher SCR amplitude to the CS+ following the US devaluation phase. This current finding is consistent with predictions that participants who have a stronger conditioned acquisition response are more likely to experience intrusions and PTSD symptoms over the week following the experiment.

Trait anxiety

Pre-trauma anxiety has been associated with PTSD development following trauma (Breslau et al., 1991; Ozer et al., 2008) and trait anxiety has been shown to be associated with the conditioned acquisition response (Mineka & Oehlberg, 2008; Zinbarg & Mohlman, 1998). When STAI-T was included as a covariate in the above analysis, the relationship between STAI-T and the dependent variables was at trend level significance, V=0.0066, $F(3,106)=2.51$, p=.062. Due to the association between trait anxiety and conditioned acquisition response, correlation analysis to check for multicollinearity was also performed and revealed no significant relationship, $r_s=.13$, p=.17. This implies that, for non-clinical participants, conditioned acquisition response may be a better predictor of intrusion frequency, intrusion distress and PTSD symptoms than trait anxiety.

Figure 8: Graphs illustrating relationship between conditioned acquisition response and outcome measures per group.
Dependent variables were log transformed prior to analysis. Raw data are displayed.



H4: summary

Results indicated that having a larger amplitude SCR to CS+ following acquisition was associated with intrusion frequency (medium effect, partial η^2=.085), intrusion distress (medium effect, partial η^2=.084), and PTSD symptomatology (medium effect, partial η^2=.075) when the effect of group was taken into account. There was still a significant effect of group and CSdiffACQ on these dependent variables when participants' trait anxiety was taken into account. The association between the dependent variables and trait anxiety was at trend level significance. This implies a significant relationship between acquiring a stronger conditioned fear response and experiencing more intrusions, more intrusion distress and more PTSD symptoms.

Hypothesis 5 (H5): Participants who have a lower score on the Response to Memories questionnaire (R2M) will experience fewer intrusions, be less distressed by them and have fewer associated PTSD symptoms in the week following the trauma paradigm.

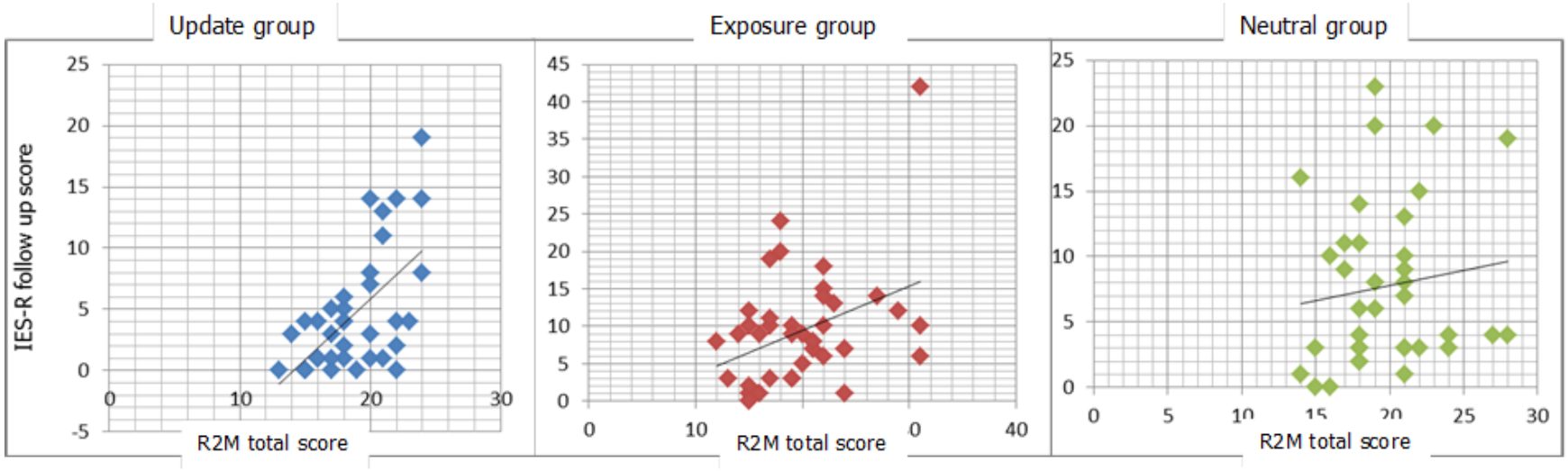
The MANCOVA conducted in H4 was re-run to include the R2M total score as a covariate. The CSdiffACQ and the effect of group remained significant and there were no significant interaction effects. This analysis revealed, using Pillai's trace, a significant relationship between the total score on the R2M and the dependent variables, V=0.14, $F(3,106)=5.84$, p=.001, partial η^2=.14. Follow-up tests revealed that R2M total score was significantly related to the IES-R follow-up scores $F(1, 108)=16.25$, p=.000, partial η^2=.13 (see Figure 9). There was no significant relationship between R2M total scores and intrusion frequency or intrusion distress. It is perhaps not surprising that there is a significant relationship between total IES-R scores and total score on the R2M questionnaire as participants completed these self-report questionnaires at the same time and some items on the IES-R are similar to items on the R2M questionnaire.

H5: summary

Analysis indicated a significant relationship between participants' self-reported responses to memories of the films and their PTSD symptomatology at follow-up. Participants who reported having more maladaptive responses to the memories of the trauma films, reported more PTSD symptoms (medium to large effect, partial η^2=.13). There was no significant relationship between intrusion frequency or intrusion distress and scores on the R2M questionnaire.

Figure 9: Graphs illustrating relationship between score on Response to Memories questionnaire and IES-R follow-up score per group.

Intrusion frequency, distress and IES-R scores were log transformed prior to analysis. Raw data are displayed.



3.3 Summary of results

There were no significant differences between the groups in terms of baseline characteristics including demographic factors and scores on anxiety, depression and trauma history questionnaires. Importantly, groups did not differ significantly in terms of SCR amplitude or distress ratings to the CS+ACQ or SCR during the acquisition films. There were also no significant differences between the groups in compliance with completing the intrusion diary. Therefore, prior to experimental manipulation groups did not differ on any of the variables measured.

In terms of H1, analysis indicated that the conditioned fear response (as measured by SCR amplitude and subjective distress) can be produced in humans in the laboratory using trauma film stimuli as the US with approximately two-thirds of participants showing a conditioned SCR above minimum response criteria. H2 investigated the effect of adding a cognitive component (update) during the US devaluation phase on physiological response and subjective distress ratings to the CS+ as compared to the exposure and neutral groups. There was a significant difference between the groups in terms of the change in the physiological conditioned fear response following US devaluation but not in the expected direction. The exposure group had a significantly greater decrease in SCR amplitude to the CS+ than the update group. Unexpectedly, the update group on average displayed an increase in the conditioned fear response following the US devaluation phase. Changes in subjective ratings of distress were in the predicted direction with the update group having a significantly larger drop in their distress ratings to the CS+ following US devaluation than both the other groups. The groups did not differ following the re-acquisition phase.

It was also investigated whether adding a cognitive component to the US devaluation phase reduced the intrusion frequency, intrusion distress and self-reported PTSD symptoms (H3). Those in the update group had significantly fewer intrusions and PTSD symptoms than the other two groups. It was predicted that participants who displayed a stronger conditioned acquisition response (SCR amplitude) and reported more maladaptive responses to the intrusions they experienced would experience more intrusions, intrusion distress and PTSD symptoms (H4 & H5). Conditioned acquisition response was significantly related to intrusion frequency, distress and PTSD symptoms when group and trait anxiety were taken into account. Participants' self-reported maladaptive responses to intrusions were significantly related to

their PTSD symptomatology but the significance of this finding is questionable due to the overlap of items in the IES-R and R2M.

In summary, these results were mostly consistent with the hypotheses. The results suggest that the fear response can be conditioned in the laboratory using trauma film stimuli as the US. Adding a cognitive component to US devaluation reduced fear conditioning, as measured by ratings of subjective distress, compared to exposure alone or watching neutral films. However, according to SCR, exposure to the US reduced the conditioned fear response more effectively than updating, and updating may actually lead to an initial increase in fear conditioning. Updating the meaning of the US led to fewer intrusions and PTSD symptoms in the week following the experiment compared to the other two groups. Conditioned acquisition response (as measured by SCR) was significantly associated with intrusion frequency, intrusion distress and PTSD symptomatology. Maladaptive responses to intrusions were linked to PTSD symptomatology. Therefore adding a cognitive component to a US devaluation process led to fewer intrusions and PTSD symptoms and reduced subjective distress to the CS+, but also led to the less effective reduction of the SCR to the CS+ than exposure.

4. Discussion

The main aim of this study was to investigate whether adding a cognitive update to a US devaluation process would reduce the conditioned fear response and PTSD symptoms compared to further exposure to the trauma films or watching neutral films. Prospective experimental studies are needed to better understand causal factors in PTSD and to develop early interventions but it is clearly unethical to expose people to real trauma. Therefore, the well established trauma-film paradigm was used.

Primarily, it was important to establish whether trauma films can produce the conditioned fear response (H1) as they have not previously been used in fear conditioning paradigms. The main hypotheses were then investigated. It was predicted that updating the meaning of the US would reduce the conditioned fear response more effectively (H2) and further reduce intrusions and analogue PTSD symptoms (H3) compared to exposure alone or neutral films. Predictors of analogue PTSD symptoms were also investigated including conditioned fear acquisition response (H4) and responses to memories of the films (H5).

4.1 Can trauma films be used as unconditioned stimuli (H1)?

This study demonstrated that trauma film stimuli (trauma films plus an *intensive presentation phase)* can be used to induce conditioned fear as measured by SCR and distress ratings. Approximately two-thirds (65%) of the cases were found to produce an SCR amplitude greater than 0.02µs to the unreinforced CS+ACQ and there was a significant difference found between participants' reactions to the CS+ACQ and CS-ACQ. This strongly implies that fear conditioning can be induced in the laboratory using trauma film stimuli as the US. The trauma paradigm was also effective in inducing intrusive memories of the films, as 95% of participants reported at least one intrusion.

In conditioning, a US is a stimulus that provokes a response before conditioning has occurred (Bouton, 2007); most recent conditioning studies have used electric shocks as the US (e.g. Kindt & Soeter, 2011; Schiller et al., 2010). Using trauma films as the US rather than single stimuli, such as a shock, has advantages but may also have conceptual difficulties. A trauma film is similar to real life trauma in that they both potentially contain multiple stimuli (USs and CSs) rather than being one single clearly defined US. This may mean that it is unclear which

part or parts of the trauma films are producing the unconditioned response and whether this is consistent across participants. On the other hand, single shocks are potentially more limited in generalising to real trauma and would be more difficult to devalue cognitively. Intrusion frequency and PTSD symptomatology can also be measured when the trauma film paradigm is used, which enables the investigation of the relationship between the conditioned fear response and analogue PTSD symptoms.

To our knowledge, this is the first study that has combined conditioned fear paradigms and the trauma film paradigm with follow-up measures. Using trauma film stimuli as the US has advantages of being a better analogue of real-life trauma, being more readily experimentally manipulated and allowing the concurrent investigation of conditioned fear and PTSD symptomatology.

4.2 Does adding a cognitive update to a US devaluation paradigm reduce the conditioned fear response (H2), intrusions and PTSD symptoms (H3)?

Participants were allocated to one of three US devaluation groups. A cumulative design was used, in an order predicted to increase the reduction of the conditioned fear response: the neutral group involved no intervention, the exposure group involved exposure to the US and the update group added a cognitive component to exposure. Both the second and third groups were designed to devalue the US through habituation to the films and through changing the meaning of the films. It was predicted that adding cognitive devaluation to exposure would further reduce the conditioned fear response.

4.2.1 Effect of experimental manipulation on fear conditioning

Surprisingly, adding a cognitive component to exposure (update group) significantly increased SCR amplitude to the CS+ following US devaluation relative to the exposure group. On average from the acquisition to US devaluation phases, the update group's SCR increased whereas the neutral and exposure groups' responses decreased. The difference between the exposure and neutral groups approached significance. In contrast, participants in the update group reported a significantly greater drop in subjective ratings of distress to the CS+ from the acquisition to US devaluation phases compared to the other two groups. This implies that, consistent with the predictions, cognitively devaluing the US reduces distress to conditioned stimuli (CS+)

more effectively than exposure to the US or no intervention. There was a drop in distress ratings in all groups following US devaluation with the largest drop in the update group, then the neutral group and finally the exposure group. This is consistent with predictions and in the opposite direction to the changes in SCR.

These results highlight inconsistency between measuring the conditioned fear response according to SCR compared to subjective distress ratings. It has been well documented that self-reported emotional experiences do not correlate well with SCR (Lang, 1985; Wilson & Keane, 2004). It has also been illustrated that a decline in the expectancy ratings of a US following the CS+ does not automatically result in a decline in SCR (Biferno & Dawson, 1977) and the CS+ can still evoke an SCR. Retrospective ratings of distress were also used in this study which may have impacted on the reliability of the distress ratings.

There are several possible explanations as to why adding a cognitive component increases the conditioned fear response according to SCR. One explanation is that exposure to the trauma films resulted in more effective US devaluation than updating the meaning. Due to the additive nature of the design, the implication is that the addition of cognitive updating to exposure reduces the effectiveness of US devaluation at test. This could be consistent with research illustrating that Psychological Debriefing can lead to the slowing of recovery (NICE, 2005; Rose et al., 2002). However, it appears that the relationship is more complicated than this as the update group reported significantly less subjective distress following US devaluation and significantly fewer intrusions and PTSD symptoms at follow-up. It may be that the new information and verbally enhanced representation of the trauma films in the update group prevents habituation of the conditioned fear response initially. This response then falls with further exposure to the CS/US pairing as seen in the re-acquisition phase. In therapy, emotional arousal is considered to be important for therapeutic change (Greenberg & Pascual-leone, 2006) and the construction of new meaning is needed for lasting changes (Whelton, 2004). Therefore, the update condition may have facilitated higher emotional arousal (i.e. SCR) alongside new information, leading to fewer intrusions and PTSD symptoms at follow-up.

A further explanation is consistent with the SCR reflecting an orienting attentional response rather than a fear arousal response. Whilst SCR has been used extensively as a measure of fear conditioning (e.g. Dibbets, Poort, & Arntz, 2012; Lissek et al., 2005; Schiller et al., 2010) and is considered to be an accurate measure of emotional arousal in carefully controlled

experimental paradigms, it can represent changes in other processes including attention and information processing (Dawson, Schell, & Filion, 2007). The update group contained new verbal information and so it may be that the participants in this group were more engaged than those in the other groups and so SCR represents a difference in how alert they were in general. However, there were no differences between the update and exposure groups in their SCR during the US devaluation films, implying that both groups experienced similar levels of arousal and attention during the films making this explanation less plausible. There is evidence to indicate that successful therapy results in an increase in attentional allocation to threat cues and it is suggested that this enables the re-appraisal of the threat cue (Adenauer et al., 2011). Therefore, the larger SCR may represent reduced attentional avoidance to the CS+ in the update group compared to the exposure group and this would also correspond with the fall in subjective ratings of distress and the reduced number of analogue PTSD symptoms.

An alternative explanation is that cognitively devaluing the US in the update group leads to the mental rehearsal of the CS/US association, leading to a slower reduction in the conditioned fear response. For example, Dibbets, Poort, & Arntz (2012) found that adding cognitive US devaluation led to the slower extinction of the CS+ as measured by expectancy ratings compared to groups receiving extinction without cognitive devaluation. Although the CS+ was not part of the devaluation procedure in this study, participants may have been rehearsing this association as they were asked to work out the meaning of the CS+ and the CS-.

Therefore, the finding that retrospective subjective levels of distress were significantly lower following US devaluation in the update group is consistent with predictions that changing the meaning of the US reduces subjective distress to the CS+. However, the significantly increased SCR to the CS+ in the update compared to the exposure group, following the US devaluation phase, was inconsistent with predictions and there are a number of explanations for this. Perhaps the explanation which is most in keeping with the other findings is that the increased SCR represents a reduction in attentional avoidance of the CS+ allowing reappraisal of the CS+.

4.2.2 Effect of experimental manipulation on intrusions and PTSD symptomatology

Participants in the update group experienced significantly fewer intrusions and PTSD symptoms (as measured by IES-R) in the week following the experiment than either the neutral or exposure groups. There are several possible explanations for this finding.

One explanation is that adding a cognitive update reduced analogue PTSD symptoms by two mechanisms: (1) altering negative appraisals of the film content and (2) enhancing verbal-conceptual processing of the trauma memory. This is consistent with cognitive models of PTSD which highlight the role of negative appraisals of the trauma (Ehlers & Clark, 2000) and the disorganised nature of the trauma memory in the development of PTSD symptoms (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000). These results also support the idea that the most effective treatments for PTSD are those that pay attention to the trauma memory and its meaning (Ehlers & Clark, 2008).

Evidence suggests that a person's appraisal of the trauma plays a crucial role in the development and maintenance of PTSD (Bryant & Guthrie, 2007; Dunmore, Clark, & Ehlers, 1999; Halligan et al., 2003) and analogue studies have illustrated that training participants to process information in a different way leads to fewer intrusions (Woud et al., 2012; White & Wild, submitted). Updating the meaning of the film may also act to enhance verbal-conceptual processing of the US and therefore interfere with the consolidation of the fear memory. According to cognitive theory (Brewin et al., 2010; Ehlers & Clark, 2000), verbally enhancing the trauma memory is predicted to reduce intrusion frequency via increased integration and contextualisation of the trauma memory allowing better top-down control. It may be that the update condition directly facilitates this or, alternatively, that the update condition reduces anxiety during the US devaluation phase enabling increased contextual processing (e.g. Leary, Adams, & Tate, 2006) of the films. This finding, therefore, lends support to the proposal that altering negative appraisals of the US and a shift towards verbal-conceptual processing may lead to a reduction in PTSD symptoms.

In terms of conditioning theory, US devaluation is predicted to lead to a reduction in intrusion frequency by changing the meaning of the fear memory underlying trauma-related symptoms (Arntz, 2011; Davey, 1989). Both the exposure and update conditions may act through US devaluation: exposure through habituation and updating by habituation and cognitively changing meaning. As updating involves exposure to the trauma memory and the inclusion of new verbal information, it may act at both the verbal level and the sensory, emotional and behavioural levels. Therefore, this study provides evidence that changing the meaning of the US may devalue the US more effectively than exposure alone.

Another explanation is that the update group may have had a higher working memory (WM) capacity load than the other two groups. Research has illustrated that participants develop fewer intrusions if WM capacity is reduced (Holmes, Brewin, & Hennessy, 2004; Holmes et al., 2009; Krans, Naring, & Becker, 2009). In this study, WM capacity in the acquisition phase was identical for all groups and the US devaluation phases were similar in terms of length of intervention. Both the update and neutral conditions contained additional information; the neutral group received new visual information whilst the update group received new verbal information. The difference may, therefore, be explained by reduced verbal WM capacity in the update group during the US devaluation phase interfering with memory consolidation processes. However, the new information was limited in terms of its WM demands and was given before the films began. A previous study with a similar design and groups concluded that these groups would have similar WM demands (Hagenaars & Arntz, 2012).

An alternative explanation is that adding an updated meaning to the films changes people's responses to the films and intrusions after the experiment e.g. they are less likely to avoid thinking about the films. This is consistent with cognitive models (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000) which suggest that strategies people use to control their sense of current threat maintain PTSD symptoms. However, there was no difference between the groups in the strategies participants reported using in response to intrusions (as measured by R2M questionnaire), making this explanation less credible.

Another explanation is that the update group had a more positive mood than the other groups when finishing the task, as low mood is linked to greater intrusion frequency (Davies & Clark, 1998b). However, there were no significant differences between the groups in terms of their overall ratings of distress or their SCR during the US devaluation films (for the update and exposure groups) implying that both groups experienced similar levels of arousal to the films. In addition, any impact on intrusion frequency from changes in mood would be short-lived (less than 24 hours) as illustrated by studies aiming to induce low mood (Hunt, 1998; Watkins, 2004) and participants in this study experienced intrusions over the entire week. Further to this, in terms of immediate mood induction, the neutral group's mood following the films might be predicted to be more positive than the update group and yet the neutral group experienced significantly more intrusions. Similar studies that have included a positive mood induction group have found that this group experience significantly more intrusions than the intervention groups (Hagenaars & Arntz, 2012). Mood induction is therefore a less likely

explanation as: the ratings of overall distress and SCR to the films did not differ between the groups, intrusions were reported across the week and the neutral group experienced significantly more intrusions than the update group.

Therefore, these findings support research highlighting that the meaning people assign to a trauma and the nature of the trauma memory are important components in the development of PTSD and that treatments targeting these two areas can reduce the likelihood of intrusions and PTSD symptoms. Encouragingly, in terms of generalising the findings, it also infers that an externally generated meaning may be effective in reducing intrusion frequency and PTSD symptoms.

4.3 What predicts analogue PTSD symptoms?

4.3.1 Conditioned acquisition response

There was a significant relationship between showing a larger conditioned acquisition response (as measured by SCR amplitude) and experiencing more intrusions, more intrusion distress and more PTSD symptoms. This is consistent with conditioning theories of PTSD which highlight the role of CS/US contingencies in the development of PTSD (Mineka & Oehlberg, 2008; Rothbaum & Davis, 2003). It also supports Ehlers & Clark's (2000) inclusion of stronger associative learning at the time of trauma to explain why people with PTSD experience a strong sense of current threat to vaguely similar cues. The results are also in keeping with previous research demonstrating that people with anxiety disorders acquire fear conditioning more strongly than those without (Lissek et al., 2005) and that differences in extinction learning may predict future PTSD development (Lommen et al., 2013). These results imply that enhanced fear conditioning may be a pre-trauma vulnerability factor for PTSD.

The relationship between enhanced fear conditioning and anxiety disorders may operate at a number of stages. For example, a stronger association may mean that the fear response is more readily triggered by cues, triggered by more loosely associated cues, is more difficult to extinguish or that the return of fear is more likely following extinction. Mineka & Oehlberg (2008) postulate that anxious individuals may produce conditioned responses with greater magnitudes leading to responses that are more difficult to extinguish or it may be that extinction alone is different in people with anxiety disorders with no difference at acquisition. This current finding is consistent with work by Orr et al. (2000) who propose that individual

differences in acquisition explain why some people develop anxiety disorders and some people do not.

Mineka & Oehlberg (2008) highlight the need for research prospectively investigating the relationship between fear conditioning and the development of anxiety disorders. To our knowledge, this is the first study to combine a conditioning paradigm with the trauma film paradigm to enable a prospective analogue design investigating how individual differences in fear conditioning impact on PTSD symptom development. This finding provides evidence that individuals with stronger conditioned acquisition responses experience more PTSD symptoms.

4.3.2 Trait anxiety

Pre-trauma anxiety is associated with the development of PTSD (Breslau et al., 1991; Ozer et al., 2008). When trait anxiety (as measured by the STAI-T) was included in the analysis, trend level significance was found and significant effects of conditioned acquisition response and group remained. People with higher trait anxiety have been shown to acquire aversive conditioning more strongly and more rapidly (Mineka & Oehlberg, 2008; Zinbarg & Mohlman, 1998). However, a check for multicollinearity revealed no significant relationship between conditioned acquisition response and STAI-T. Therefore, these results imply that, in the non-clinical population, conditioned acquisition response is a better predictor of intrusion frequency, intrusion distress and PTSD symptoms than trait anxiety.

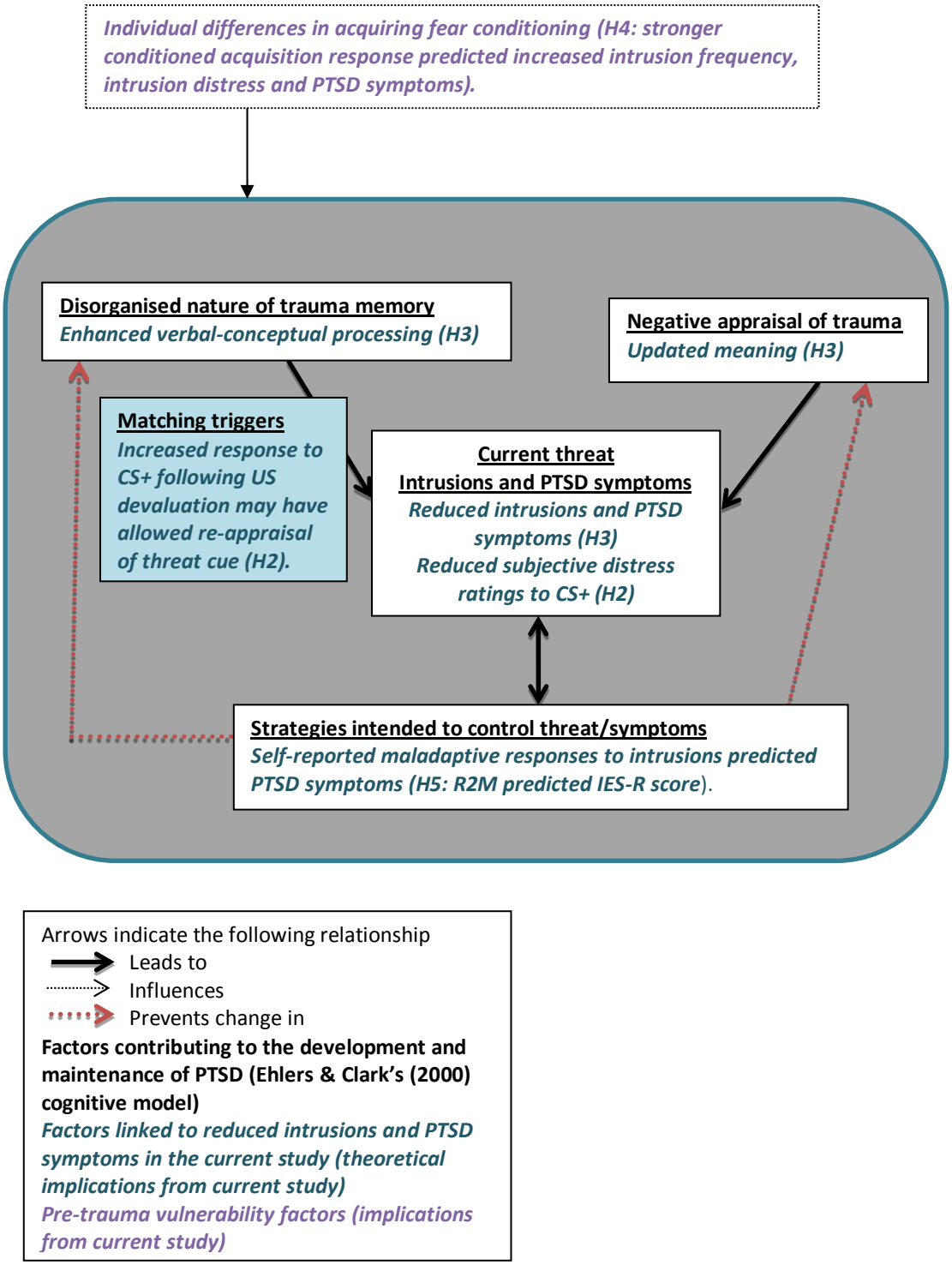
4.3.3 Response to memories of the films

There was a significant relationship between self-reported maladaptive responses to intrusions (as measured by the R2M questionnaire) and self-reported PTSD symptoms (as measured by the IES-R). This is in keeping with studies that have shown the development and maintenance of PTSD is linked with maladaptive coping styles such as rumination and thought suppression (Clohessy & Ehlers, 1999; Dunmore, Clark, & Ehlers, 1999; Mayou, Ehlers, & Bryant, 2002; Murray, Ehlers, & Mayou, 2002). It is also consistent with cognitive models of PTSD which postulate that PTSD symptoms can be maintained by maladaptive coping strategies, for example avoidance of the memory preventing the integration and contextualisation of the trauma memory (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000). However, there is overlap between items on the IES-R and R2M making this finding unsurprising and, contrary to predictions, there was not a significant relationship between intrusion frequency and R2M.

4.4 Theoretical implications

The results are in keeping with cognitive models of PTSD that predict: (1) changing negative appraisals of the trauma films (Ehlers & Clark, 2000) and (2) enhancing verbal-conceptual processing of the trauma memory leads to fewer intrusions and PTSD symptoms compared to controls (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000). The update group aimed to target both of these processes and the significant difference in intrusion frequency and PTSD symptoms between the update and other two groups supports the role of these processes in the development of PTSD. An increased response to associated stimuli (CS+) in the update group following US devaluation may represent reduced attentional avoidance facilitating re-appraisal of the cue. Another important finding with theoretical connotations is that conditioned SCR acquisition response predicted intrusion frequency, distress and PTSD symptoms. This finding lends support to conditioning theories of PTSD and may explain, in part, individual differences in PTSD development. This finding implies that acquiring fear conditioning more strongly is a vulnerability factor for PTSD. The findings are summarised in Figure 10 in terms of how they relate to Ehlers & Clark's (2000) cognitive model.

Figure 10: findings from current study embedded in Ehlers & Clark's (2000) cognitive model



Both Brewin, Dalgleish, & Joseph (1996) and Ehlers & Clark (2000) highlight that a shift from verbal-conceptual processing to visuospatial perceptual processing at the time of trauma results in a disorganised and poorly integrated memory, rich in sensory detail and experienced as if the trauma is occurring again. Cognitive models predict that a shift away from visuospatial processing leads to fewer intrusions and disrupting verbal processing leads to more. This has been demonstrated using the trauma film paradigm (Deeprose et al., 2012; Holmes, Brewin, & Hennessy, 2004; Holmes et al., 2009; Stuart, Holmes, & Brewin, 2006). Cognitive models also predict that verbal enhancement should lead to a reduction in intrusions but studies have failed to evidence this (Holmes, Brewin, & Hennessy, 2004; Krans, Naring, & Becker, 2009). Holmes & Bourne (2008) suggest that this may be because participants were not engaged enough with the emotional meaning of the films to shift processing towards verbal-conceptual processing. In contrast to DRT, general memory and attention theory would predict no significant differences between a verbal enhancement and exposure group (Krans, Naring, & Becker, 2009). This current study may be the first to demonstrate that verbally enhancing the memory leads to fewer intrusions and PTSD symptoms as predicted by DRT (Brewin, Dalgleish, & Joseph, 1996; Brewin et al., 2010) despite levels of fear and arousal during the films (as measured by SCR) being the same for the two intervention groups. The update group may be able to integrate the trauma memory more fully through increased verbal-conceptual processing leading to fewer intrusions due to better top-down control.

A theoretical implication of the significant difference between the neutral and update groups in analogue PTSD symptoms is the importance of combining the activation of the trauma memory (update and exposure groups) with altering the meaning (update group). The neutral group watched films with related content and so, if participants only needed to learn that there can be innocuous consequences to, for example, driving you would predict that the neutral group would also experience fewer intrusions. Therefore, neither activating and habituating to the trauma (exposure group) nor learning new information about related stimuli (neutral group e.g. driving) is sufficient to reduce analogue trauma symptoms. Combining activation of the memory and new information is consistent with updating procedures in TF-CBT where people with PTSD recount the details of the trauma and incorporate new information. Emotional Processing Theory highlights the importance of activating the fear structure before incorporating new information (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989).

The finding that conditioned SCR amplitude was significantly larger following US devaluation for the update group compared to the exposure group, coupled with the finding that the update group had significantly fewer analogue PTSD symptoms has interesting theoretical and clinical implications and requires replication. As discussed above, there are a number of possible reasons why the update group had a significantly larger SCR than the exposure group. One theoretical implication is that cognitive US devaluation has a different effect on fear conditioning than devaluation by exposure/habituation and that there is not a simple cumulative relationship of adding a cognitive component to exposure. In line with a previous study (Dibbets, Poort, & Arntz, 2012), changing the meaning of the trauma films may have led to an initial slowing in the reduction of the conditioned fear response. Alternatively, it could be that increased emotional arousal and/or attentional allocation to threat cues in the update group facilitates information processing and integration of trauma memories, reducing PTSD symptoms.

4.5 Clinical implications

4.5.1 Early intervention

Whether treatments that target the meaning of the trauma are more effective than exposure alone or more effective in combination is hotly debated and mixed results have been reported. For early intervention, some suggest comparable results (Shalev et al., 2012) whilst others suggest that exposure, rather than cognitive restructuring, is the key component in preventing ASD developing into PTSD and increasing functioning following trauma (Bryant et al., 2008; Bryant et al., 1999). In terms of therapy for chronic PTSD, some studies have indicated similar outcomes for CBT and exposure therapy (e.g. Paunovic & Ost, 2001), whilst others have indicated that CBT may be more effective than imaginal exposure in the long term (at five-year follow-up, Tarrier & Sommerfield, 2004) but not at 12-month follow-up (Tarrier et al., 1999).

These current findings support the assertion that the most effective treatments for PTSD are those focusing on the person's trauma memory and its meaning (Ehlers & Clark, 2008) and support the addition of cognitive techniques to exposure. Updating shortly after the trauma may target the consolidation of the trauma memory and disrupt its storage. It may be effective by jointly targeting the appraisal of the trauma and increasing verbal-conceptual processing of the trauma memory. These results imply that the addition of an intervention at the verbal-conceptual level is important and may be an exciting tool to prevent the development of PTSD

symptoms. However, the findings also highlight that there is not a straightforward relationship between adding a cognitive updating component, the fear response (as measured by SCR and subjective distress ratings) and the development of PTSD symptoms.

Interventions for occupational groups

These findings could be used to inform training programmes for at-risk occupational groups. Prevalence of PTSD has been found to be elevated in some occupational groups compared to the general population e.g. ambulance workers (Sterud, Ekeberg, & Hem, 2006). Training programmes would need to be carefully developed for each occupational group and may involve highlighting the importance of processing conceptual aspects of the trauma, verbally updating the memory, being aware of their own negative appraisals and skills to challenge these. This could also be used as a prevention strategy as previous studies have illustrated that training people in an appraisal style can reduce intrusion frequency (Woud et al., 2012; White & Wild, submitted).

One concern might be that changing the way in which occupational groups perform their job may interfere with their competency or efficiency. For example, it may be detrimental for an ambulance worker to focus on the conceptual rather than perceptual details of an emergency. This study provides evidence that intervention *after* analogue trauma can be effective in reducing PTSD symptoms. Therefore, it might be that workers are trained to enhance verbal-conceptual processing of the trauma and attend to any negative appraisals in the consolidation period following a difficult event rather than during the event itself. However, given the limited ability to generalise analogue trauma films to real-life trauma, this would need to be carefully thought through and researched for each occupational group. This may be linked to research supporting the use of group-based psycho-education targeting at-risk occupational groups, such as military personnel (Adler et al., 2009) .

4.5.2 Therapeutic process

Interestingly, the update group had both a stronger conditioned fear response following US devaluation and reported fewer intrusions and PTSD symptoms than the exposure group. Clinical implications for this finding are tentatively suggested which include the importance of emotional arousal and reducing attentional avoidance for therapeutic change. These two processes are complementary as reduced attentional avoidance of the threat cue (CS+) would

be associated with increased arousal and this combination may enable greater processing and reduced PTSD symptoms.

Recovery from anxiety disorders is thought to be reliant on the person actively experiencing their anxiety whilst confronting their fear. For example, positive outcomes for PTSD are associated with the expression of fear during the first exposure session (Foa et al., 1995; Jaycox, Foa, & Morral, 1998), early elevations in psychophysiology during imaginal flooding predict improvements in intrusions (Pitman et al., 1996) and outcomes for people with OCD are predicted by the level of anxiety activation during exposure (as measured by self-report, cardiac response and EDA) (Kozak, Foa, & Steketee, 1988). Beck (1996) hypothesises that the presence of strong affect can make changes in cognitions more effective and durable and that cognitions are most malleable when the person is emotionally aroused (Hunt, 1998). The combination of emotional arousal and perceptual processing during therapy has also been shown to better predict reductions in depressive and psychopathological symptoms than either alone (Missirlian et al., 2005). Reviews have concluded that effective therapy requires emotional engagement and arousal and that the construction of new meaning is needed for durable change (Greenberg & Pascual-leone, 2006; Whelton, 2004). Moderate emotional arousal has been shown to be optimal for therapeutic change, with the suggestion that high emotional arousal may disrupt cognitive processing and emotional regulation (Carryer & Greenberg, 2010). Therefore, it is tentatively suggested that the updating condition may facilitate moderate emotional arousal to trauma-related cues (as indicated by SCR to the CS+USDe).

Research has illustrated that aversive cues initially evoke a rapid response and then people use attentional avoidance to reduce the sense of current threat in the short term (Koster et al., 2005). Therapy (Narrative Exposure Therapy, NET) may lead to an increase in attentional allocation to potential threat cues, enabling the person to re-appraise the current threat and so reduce PTSD symptoms (Adenauer et al., 2011). Adenauer et al. (2011) proposes that NET enhances voluntary top-down episodic memory search to a potentially threatening cue, allowing the person to evaluate the current threat of the stimulus based on previous memory traces. This voluntary top-down search would be in keeping with DRT (Brewin, Dalgleish, & Joseph, 1996) which proposes that therapy enhances the elaboration and integration of C-reps, enabling top-down control of the lower level S-reps. Cognitive models of PTSD (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000) highlight a central role of avoidance in

maintaining PTSD symptoms. SCR is linked to attentional focus and so it may be that, following update, increased attention is allocated to the threat cues (illustrated by the increase in SCR) allowing re-appraisal and reduction in PTSD symptoms at follow-up. It would be important to carry out this study again and cross-validate the findings with startle responses which are more sensitive to the conditioned fear response (e.g. Kindt & Soeter, 2011; Weike, Schupp, & Hamm, 2007). Either the combination of reduced attentional avoidance and increased arousal, or one process alone, may help to explain the findings of increased SCR combined with reduced distress ratings following US devaluation and reduced intrusions and PTSD symptoms at follow-up for the update compared to the other groups. Clinically, this may highlight the importance of reducing avoidance of threat cues and increasing emotional processing.

4.5.3 Identifying those at risk of developing PTSD

These findings support research highlighting that individual differences in fear conditioning may be a vulnerability factor for PTSD development. This could aid identification both of (1) individuals likely to develop PTSD following trauma and (2) individuals more likely to develop PTSD in groups regularly exposed to trauma e.g. policemen and military personnel. Previous studies have highlighted that there may be sub-types of PTSD based on the ability to acquire fear conditioning, and these different subtypes may respond differently to pharmacological intervention (Aikins et al., 2011). This indicates that fear conditioning may be a "biomarker" for PTSD development and treatment response but this requires further investigation. Therefore, psychophysiological assessment may enhance identification of those at risk and the targeting of intervention and prevention strategies towards individuals more likely to benefit.

4.6 Limitations

There were several limitations to this research. A major limitation is the degree to which the findings can generalise to people who are exposed to real-life trauma and develop PTSD, since the study used non-clinical participants with films as the analogue trauma. How comparable experimental fear conditioning and US devaluation or extinction studies are with anxiety disorders and therapeutic techniques is difficult to establish. Anxiety can be considered to be more complex with greater individual variation than fear; fear may represent a basic human emotion with evolutionary adaptive functions whilst anxiety may incorporate additional cognitive components to associate basic emotions to meaning and context (Hofmann, 2009;

Izard, 1992). However, both represent functional responses to threat that, if they become excessive, can lead to anxiety disorders (Hofmann, 2009).

In therapy, exposure and updating techniques are more complex than the analogue interventions used in this study. This study externally generated the updated meaning whilst, in therapy, time would be spent generating a subjective meaning with the patient and the techniques would be carried out until distress ratings are low rather than after a fixed number of trials. However, the experimental interventions were generated based on those used in evidence-based therapy and the aims of both experimental techniques were in keeping with therapy, i.e. updating aimed to add detail and change meaning and exposure to habituate to the fear stimuli. This limitation therefore partly reflects the discrepancy between attempting to carefully control intervention techniques in experimental paradigms and the collaborative and tailored nature of therapeutic interventions. Further to this, the acquisition/US devaluation/re-acquisition phases were carried out consecutively which does not reflect real life and the effectiveness of the US devaluation and re-acquisition phases may have been enhanced if there was a greater time delay between the stages.

A further limitation is that the nature of the recruitment process may have skewed the sample. The participants were self-selected and, due to ethical constraints, clear information was given to participants about film content prior to volunteering. Therefore, volunteers may be people who are less distressed by watching trauma films than the average population. In addition, participants had chosen to take part and the expectancy that they were going to watch trauma films may have meant that their sense of control and arousal levels were not in keeping with people who experience real-life trauma.

This study used retrospective subjective ratings of distress, as is commonly done in conditioning studies (Soeter & Kindt, 2011; Vansteenwegen et al., 2005; Vervliet, Vansteenwegen, & Eelen, 2004). However, retrospective reporting makes the ratings vulnerable to distortion. This was done to avoid interfering with the natural development of electrodermal activity and due to practical limitations in the set-up of the psychophysiological equipment meaning that the procedure could not be paused. Validity of these ratings is increased by there being no difference in baseline distress ratings between the groups and that the ratings followed the pattern expected from previous research (Dibbets, Poort, & Arntz, 2012; Schiller et al., 2010).

Another limitation is the non-specific nature of SCR and this study would be enhanced by having multiple indexes of fear conditioning, such as startle responses and US expectancy ratings (Kindt & Soeter, 2011). SCR has been used extensively as a measure of fear conditioning in humans (e.g. Milad et al., 2005; Schiller et al., 2010; Vansteenwegen et al., 2005; Vervliet, Vansteenwegen, & Eelen, 2004). However, it is a non-specific measurement of arousal that may index attentional processes associated with the orienting response (Dawson, Schell, & Filion, 2007; Filion et al., 1991). If SCR is a measure of attention then, as fear-relevant stimuli draw a preferential and more prominent attentional focus (Carretié et al, 2001; Öhman, Flykt, & Esteves, 2001), it would be predicted that SCR co-varies with anxious arousal (Lissek et al., 2005). Limitations to SCR are that it may reflect anticipatory arousal regardless of the valence of the CS+ (Hamm & Vaitl, 1996) and it may be dependent on being aware of the CS/US association (e.g. Klucken et al., 2009; Tabbert et al., 2006). Cross-validation using the startle response would have been helpful as it may be a more reliable indicator of fear (Kindt & Soeter, 2011). Expectancy ratings may not have been as appropriate in this study as it investigated US devaluation, which would be predicted to have an impact on distress and SCR but not necessarily the expectancy of the US following the CS+. In addition, since SCR is sensitive to attentional processes (Filion et al., 1991), expectancy ratings may have interfered with the measurement of SCR by further directing attention to the CS/US contingency (Kindt & Soeter, 2011; Soeter & Kindt, 2011). Practical constraints meant that startle response could not be used in this study.

4.7 Future research

As this study appears to be the first to have combined conditioning and trauma film paradigms, the results require replication. The exact mechanism by which the update group experienced fewer PTSD symptoms is unclear and future studies could add tighter control groups. If the results are replicated and experimental effect is established, a study using cognitive behavioural techniques as an early preventative intervention in real-life settings (such as A&E) would be warranted to establish whether updating techniques can further limit the development of PTSD. This would be an expansion of the Rothbaum et al. (2008) study which found that exposure was an effective early intervention to reduce depression and post-traumatic reactions. It would be interesting to repeat the Rothbaum et al. (2008) study with

the addition of an cognitive update group to see whether this would enhance effectiveness further.

Further studies investigating whether increased conditioned fear acquisition predicts the development of PTSD in experimental settings, in at-risk groups prior to trauma exposure and in settings targeting people shortly after trauma exposure (e.g. A&E), would help to determine whether this could be a reliable index for identifying people vulnerable to PTSD development. Further to this, it would be useful to expand on pilot studies investigating whether the acquisition response is associated with treatment outcome (Aikins et al., 2011). For example, it might be predicted that those who show a stronger acquisition response are more likely to develop PTSD and are therefore more likely to derive therapeutic gain from early intervention. In addition, further research into the relationship between acquisition, US devaluation/extinction and PTSD development is warranted.

Future studies could add control groups to elucidate the mechanisms that may be reducing intrusions and PTSD symptoms in the update group. For example, it may be that the update group experienced fewer intrusions due to changing the meaning of the films, that the addition of new *verbal* information disrupted consolidation or it may be the combination that has therapeutic benefit. Ehlers & Clark's (2000) cognitive model of PTSD would predict that targeting both the disorganised nature of the trauma memory and the negative appraisals of the trauma are helpful in reducing PTSD symptoms. It would be interesting to attempt to experimentally separate these processes to establish relative effect. A control group that receives additional neutral verbal information to enhance verbal processing without changing meaning compared to the update group from this study would further understanding. However, previous research indicates that enhancing verbal processing by adding detail alone is not sufficient to reduce intrusion frequency (Holmes & Bourne, 2008) pointing to the importance of changing meaning. There are other control groups that could be added to further tease apart the mechanisms and aid the development of effective early intervention. Additional control groups might include (1) neutral consequences compared to positive consequences (i.e. finding out what happened versus happy ending), (2) visually compared to verbally updating the trauma film and (3) receiving the verbal update alone without exposure.

Future studies could also expand on these findings by using fear–relevant stimuli to better model anxiety disorders (Kindt & Soeter, 2011). This study used fear-irrelevant stimuli as the

CS+ because fear–relevant stimuli are more resistant to extinction (e.g. Mineka & Öhman, 2002) and previous similar studies have failed to show a reduction in the conditioned fear response (Dibbets, Poort, & Arntz, 2012). Use of fear-irrelevant CS+s have also been recommended by previous research to allow for separate US devaluation from the CS+ (Dibbets, Poort, & Arntz, 2012). Therefore, fear-irrelevant stimuli were used to increase the likelihood that the conditioned fear response would be successfully reduced and have been used in previous research (e.g. Schiller et al., 2010) but the impact of using fear-relevant stimuli should be investigated.

It would be important to investigate whether varying the time between acquisition and US devaluation has an impact on treatment effect. Some studies have included a thirty minute delay between trauma films and the intervention, to mirror average waiting times in emergency departments (e.g. Holmes et al., 2009). Based on previous research, it is assumed that an intervention taking place in the consolidation window (approximately six hours) may be more effective in re-writing the trauma memory (Holmes et al., 2009; Schiller et al., 2010). However, further investigation is warranted into whether effectiveness changes both within this six-hour window and outside it.

4.8 Conclusions

The findings were consistent with the majority of the hypotheses. The trauma film stimuli were able to produce the conditioned fear response supporting their use as unconditioned stimuli. Adding a cognitive update to a US devaluation process reduced (a) fear conditioning as measured by subjective distress ratings and (b) intrusions and PTSD symptoms at follow-up. Participants that had a stronger conditioned acquisition response reported more intrusions, more intrusion distress and more PTSD symptoms. However, in contrast to hypotheses, this study suggests that adding a cognitive update increased conditioned SCR following US devaluation.

The finding that the update group reported significantly reduced subjective distress to the CS+ following US devaluation and reported significantly fewer intrusions and PTSD symptoms is consistent with cognitive models of PTSD highlighting that the meaning of the trauma (Ehlers & Clark, 2000) and the nature of the trauma memory are important in the development of PTSD (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000). A shift in processing style from

verbal to visuospatial during trauma is predicted to lead to re-experiencing symptoms and this study provides evidence that enhancing verbal processing can reduce intrusions and PTSD symptoms. This supports further research into adding cognitive components, focussing on the memory and its meaning, to early intervention and training programmes for at-risk groups.

The increase in conditioned SCR following US devaluation in the update group may be consistent with research highlighting the importance of emotional arousal in therapy and increased attentional allocation to threat cues following therapy. Clinically, this may have implications for the therapeutic process but requires further investigation and replication. This finding also highlights the discrepancy between measuring the conditioned fear response using SCR and subjective distress ratings.

The finding that conditioned acquisition response predicted intrusion frequency, distress and PTSD symptoms lends support to conditioning theories proposing that individual differences in conditioned fear acquisition explain why some people develop PTSD and some do not (Orr et al., 2000). These findings highlight that the conditioned acquisition response may be a pre-trauma vulnerability factor for PTSD. Psychophysiological assessment may aid identification of those at risk of PTSD development before and after trauma and those that may benefit more from early intervention.

This study appears to be the first to combine conditioning and the trauma film paradigms. It is also the first to investigate the additive effect of cognitive techniques on exposure compared to a neutral group using the trauma film paradigm. This study was experimental in design, had several limitations and replication is required. Whilst the findings have promising theoretical and clinical implications, whether they generalise to real-life trauma, PTSD and therapy is unclear and cross-validation with startle responses and replication with tighter control groups is warranted.

5. References

Acheson, D. T., Gresack, J. E., & Risbrough, V. B. (2012). Hippocampal dysfunction effects on context memory: possible etiology for posttraumatic stress disorder. *Neuropharmacology*, *62*(2), 674–85.

Adenauer, H., Catani, C., Gola, H., Keil, J., Ruf, M., Schauer, M., & Neuner, F. (2011). Narrative exposure therapy for PTSD increases top-down processing of aversive stimuli- evidence from a randomized controlled treatment trial. *BMC neuroscience*, *12*(127), 1–13.

Adler, A. B., Bliese, P. D., McGurk, D., Hoge, C. W., & Castro, C. A. (2009). Battlemind debriefing and battlemind training as early interventions with soldiers returning from Iraq: Randomization by platoon. *Journal of Consulting and Clinical Psychology*, *77*(5), 928–40.

Aikins, D. E., Jackson, E. D., Christensen, A., Walderhaug, E., Afroz, S., & Neumeister, A. (2011). Differential conditioned fear response predicts duloxetine treatment outcome in male veterans with PTSD: a pilot study. *Psychiatry research*, *188*(3), 453–5.

Alberini, C. M. (2005). Mechanisms of memory stabilization: are consolidation and reconsolidation similar or distinct processes? *Trends in Neurosciences*, *28*(1), 51–6.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th edn).* Washington, DC: Author.

Arntz, A. (2011). Imagery Rescripting for Personality Disorders. *Cognitive and Behavioral Practice*, *18*(4), 466–481.

Arntz, A., Tiesema, M., & Kindt, M. (2007). Treatment of PTSD: a comparison of imaginal exposure with and without imagery rescripting. *Journal of Behavior Therapy and Experimental Psychiatry*, *38*(4), 345–70.

Beck, A. T. (1996). Beyond belief: a theory of modes, personality, and psychopathology. In Salkovskis, P.M. (Ed.). *Frontiers of Cognitive Therapy*, (pp. 1–25), New York: Guilford Press.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting & Clinical Psychology*, *56*, 893–897.

Beck, J. G., Grant, D. M., Read, J. P., Clapp, J. D., Coffey, S. F., Miller, L. M., & Palyo, S. A. (2008). The Impact of Event Scale –Revised: Psychometric properties in a sample of motor vehicle accident survivors. *Journal of Anxiety Disorders*, *22*(2), 187–198.

Berntsen, D. (1996). Involuntary Autobiographical Memories. *Applied Cognitive Psychology*, *10*, 435–454.

Berntsen, D. (2001). Involuntary memories of emotional events: do memories of traumas and extremely happy events differ? *Applied Cognitive Psychology*, *15*(7), S135–S158.

Berntsen, D., & Hall, N. M. (2004). The episodic nature of involuntary autobiographical memories. *Memory & Cognition*, *32*(5), 789–803.

Biferno, M. A., & Dawson, M. E. (1977). The onset of contingency awareness and electrodermal classical conditioning: an analysis of temporal relationships during acquisition and extinction. *Psychophysiology*, *14*(2), 164–71.

Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder: Systematic review and meta-analysis. *The British Journal of Psychiatry*, (190), 97–104.

Bisson, J. I., Jenkins, P. L., Alexander, J., & Bannister, C. (1997). Randomised controlled trial of psychological debriefing for victims of acute burn trauma . *British Journal of Psychiatry*, *171*, 78–81.

Blake, D., Weathers, F., Nagy, L., Kaloupek, D., Klauminzer, G., Charney, D., & Keane, T. (1990). *Clinician-Administered PTSD Scale (CAPS)*. National Center for Post-Traumatic Stress Disorder, Behavioral Science Division Boston-VA, Boston, MA.

Blanchard, E. B., Hickling, E. J., Galovski, T., & Veazey, C. (2002). Emergency room vital signs and PTSD in a treatment seeking sample of motor vehicle accident survivors. *Journal of Traumatic Stress*, *15*(3), 199–204.

Blechert, J., Michael, T., Vriends, N., Margraf, J., & Wilhelm, F. H. (2007). Fear conditioning in posttraumatic stress disorder: evidence for delayed extinction of autonomic, experiential, and behavioural responses. *Behaviour Research and Therapy*, *45*(9), 2019–33.

Bomyea, J., Risbrough, V., & Lang, A. J. (2012). A consideration of select pre-trauma factors as key vulnerabilities in PTSD. *Clinical Psychology Review*, *32*, 630–641.

Boucsein, W., Fowles, D. C., Grimnes, S., Ben-Shakhar, G., Roth, W. T., Dawson, M. E., & Filion, D. L. (2012). Publication recommendations for electrodermal measurements. *Psychophysiology*, *49*(8), 1017–1034.

Boudewyns, P., & Hyer, L. (1990). Physiological Response to Combat Memories and Preliminary Treatment Outcome in Vietnam Veteran PTSD Patients Treated with Direct Therapeutic Exposure. *Behavior Therapy*, *21*, 63–87.

Bouton, M. E. (2002). Context, Ambiguity, and Unlearning: Sources of Relapse after Behavioral Extinction. *Biological Psychiatry*, *52*, 976–986.

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning & Memory*, *11*(5), 485–94.

Bouton, M. E. (2007). *Learning and behavior: A contemporary synthesis.* Sinauer Associates.

Bouton, M. E., & Bolles, R. (1979). Contextual Control of the Extinction of Conditioned Fear. *Learning and Motivation*, *10*, 445–466.

Breslau, N., Davis, G. C., Andreski, P., & Peterson, E. (1991). Traumatic Events and Posttraumatic Stress Disorder in an Urban Population of Young Adults. *Archives of General Psychiatry*, *48*, 216–222.

Breslau, N., Kessler, R. C., Chilcoat, H. D., Schultz, L. R., Davis, G. C., & Andreski, P. (1998). Trauma and Posttraumatic Stress Disorder in the Community. *Archives of General Psychiatry*, *55*, 626–632.

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, *68*(5), 748–766.

Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A Dual Representation Theory of Posttraumatic Stress Disorder. *Psychological Review*, *103*(2), 670–686.

Brewin, C. R., Gregory, J. D., Lipton, M., & Burgess, N. (2010). Intrusive Images in Psychological Disorders: Characteristics, Neural Mechanisms, and Treatment Implications. *Psychological Review*, *117*(1), 210 –232.

Brewin, C. R., & Holmes, E. A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review*, *23*, 339 – 376.

Brunello, N., Davidson, J. R., Deahl, M., Kessler, R. C., Mendlewicz, J., Racagni, G., Shalev, A. Y., et al. (2001). Posttraumatic stress disorder: diagnosis and epidemiology, comorbidity and social consequences, biology and treatment. *Neuropsychobiology*, *43*(3), 150–62.

Bryant, R A, & Guthrie, R. M. (2007). Maladaptive self-appraisals before trauma exposure predict posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *75*(5), 812–5.

Bryant, R. A., & Harvey, A. G. (1995). Avoidant coping style and post-traumatic stress following motor vehicle accidents. *Behaviour Research and Therapy*, *33*(6), 631–635.

Bryant, R. A., Mastrodomenico, J., Felmingham, K. L., Hopwood, S., Kenny, L., & Kandris, E. (2008). Treatment of Acute Stress Disorder: A Randomized Controlled Trial. *Archives of General Psychiatry*, *65*(6), 659–667.

Bryant, R. A., Sackville, T., Dang, S. T., Moulds, M., & Guthrie, R. (1999). Treating Acute Stress Disorder: An Evaluation of Cognitive Behavior Therapy and Supportive Counseling Techniques. *American Journal of Psychitary*, *156*(11), 1780–1786.

Buckley, T. C., Blanchard, E. B., & Neill, W. T. (2000). Information Processing and PTSD: a Review of the Empirical Literature. *Clinical Psychology Review*, *28*(8), 1041–1065.

Bugg, A., Turpin, G., Mason, S., & Scholes, C. (2009). A randomised controlled trial of the effectiveness of writing as a self-help intervention for traumatic injury patients at risk of developing post-traumatic stress disorder. *Behaviour Research and Therapy*, *47*(1), 6–12.

Butler, G., Wells, A., & Dewick, H. (1995). Differential Effects of Worry and Imagery After Exposure to a Stressful Stimulus: A Pilot Study. *Behavioural and Cognitive Psychotherapy*, *23*, 45–56.

Candel, I., & Merckelbach, H. (2004). Peritraumatic Dissociation as a Predictor of Post-traumatic Stress Disorder: A Critical Review. *Comprehensive Psychiatry*, *45*(1), 44–50.

Carretié, L., Mercado, F., Tapia, M., & Hinojosa, J. A. (2001). Emotion, attention, and the "negativity bias", studied through event-related potentials. *International Journal of Psychophysiology*, *41*, 75–85.

Carryer, J. R., & Greenberg, L. S. (2010). Optimal levels of emotional arousal in experiential therapy of depression. *Journal of Consulting and Clinical Psychology*, *78*(2), 190–9.

Clohessy, S., & Ehlers, A. (1999). PTSD symptoms , response to intrusive memories and coping in ambulance service workers. *British Journal of Clinical Psychology*, *38*, 251–265.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences (2nd edn).* Hillsdale, NJ: Erlbaum.

Conrad, C. D. (2008). Chronic stress-induced hippocampal vulnerability: the glucocorticoid vulnerability hypothesis. *Reviews in the Neurosciences*, *19*(6), 395–411.

Craske, M. G, Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, *46*(1), 5–27.

Craske, M.G. (1999). *Anxiety Disorders: Psychological Approaches to Theory and Treatment.* Westview Press, Boulder.

Creamer, M., Bell, R., & Failla, S. (2003). Psychometric properties of the Impact of Event Scale — Revised. *Behaviour Research and Therapy*, *41*, 1489–1496.

Dalgleish, T., & Yiend, J. (2006). The effects of suppressing a negative autobiographical memory on concurrent intrusions and subsequent autobiographical recall in dysphoria. *Journal of Abnormal Psychology*, *115*(3), 467–73.

Davey, G. C. L. (1989). UCS Revaluation and Conditioning Models of Acquired fears. *Behaviour Research and Therapy*, *27*(5), 521–528.

Davidson, J. R. T., Hughes, D., Blazer, D. G., & George, L. K. (1991). Post-traumatic stress disorder in the community: an epidemiological study. *Psychological Medicine*, *21*, 713–721.

Davies, M. I., & Clark, D. M. (1998a). Thought suppression produces a rebound effect with analogue post-traumatic intrusions. *Behaviour Research and Therapy*, *36*, 571–82.

Davies, M. I., & Clark, D. M. (1998b). Predictors of Analogue Post-Traumatic Intrusive Cognitions. *Behavioural and Cognitive Psychotherapy*, *26*, 303–314.

Dawson, M. E., Schell, A. M., & Filion, D. L. (2007). The Electrodermal System. In Cacioppo, J. T. Tassinary, L. G. Berntson, G. (Eds.). *Handbook of Psychophysiology* (pp. 159–181. Cambridge University Press.).

Deeprose, C., Zhang, S., Dejong, H., Dalgleish, T., & Holmes, E. A. (2012). Imagery in the aftermath of viewing a traumatic film: Using cognitive tasks to modulate the development of involuntary memory. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(2), 758–764.

Delahanty, D. L., Raimonde, A. J., & Spoonster, E. (2000). Initial posttraumatic urinary cortisol levels predict subsequent PTSD symptoms in motor vehicle accident victims. *Biological Psychiatry*, *48*(9), 940–7.

Delgado, M. R., Olsson, A., & Phelps, E. A. (2006). Extending animal models of fear conditioning to humans. *Biological Psychology*, *73*, 39–48.

Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. *Behavioral science*, *19*(1), 1–15.

Dibbets, P., Poort, H., & Arntz, A. (2012). Adding imagery rescripting during extinction leads to less ABA renewal. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(1), 614–24.

Doyère, V., Debiec, J., Monfils, M.-H., Schafe, G. E., & LeDoux, J. E. (2007). Synapse-specific reconsolidation of distinct fear memories in the lateral amygdala. *Nature Neuroscience*, *10*(4), 414–6.

Dunmore, E., Clark, D. M., & Ehlers, A. (1999). Cognitive factors involved in the onset and maintenance of posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy*, *37*, 809–829.

Durham, T. W., McCammon, S. L., & Allison, E. J. (1985). The psychological impact of disaster on rescue personnel. *Annals of Emergency Medicine*, *14*(7), 664–8.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*, 319–345.

Ehlers, A., & Clark, D. M. (2008). Post-traumatic stress disorder: The development of effective psychological treatments. *Nordic Journal of Psychiatry*, *62*.

Ehlers, A., Clark, D. M., Dunmore, E., Jaycox, L., Meadows, E., & Foa, E. B. (1998). Predicting response to exposure treatment in PTSD: the role of mental defeat and alienation. *Journal of Traumatic Stress*, *11*(3), 457–71.

Ehlers, A., Clark, D. M., Hackmann, A., Mcmanus, F., & Fennell, M. (2005). Cognitive therapy for post-traumatic stress disorder: development and evaluation. *Behaviour Research and Therapy*, *43*, 413–431.

Ehlers, A., Hackmann, A., & Michael, T. (2004). Intrusive re-experiencing in post-traumatic stress disorder: Phenomenology, theory and therapy. *Memory*, *12*(4), 403–415.

Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of abnormal psychology*, *107*(3), 508–19.

Ehlers, A., Mayou, R. A., & Bryant, B. (2003). Cognitive predictors of posttraumatic stress disorder in children: results of a prospective longitudinal study. *Behaviour Research and Therapy*, *41*(1), 1–10.

Ehlers, A., & Steil, R. (1995). Maintenance of Intrusive Memories in Posttraumatic Stress Disorder: A Cognitive Approach. *Behavioural and Cognitive Psychotherapy*, *23*, 217–249.

Ehlers, A., Suendermann, O., Boellinghaus, I., Vossbeck-elsebusch, A., Gamer, M., Briddon, E., Walwyn, M., et al. (2010). Heart rate responses to standardized trauma-related pictures in acute posttraumatic stress disorder. *International Journal of Psychophysiology*, *78*, 27–34.

Field, A. (2006). Is conditioning a useful framework for understanding the development and treatment of phobias? *Clinical Psychology Review*, *26*(7), 857–75.

Field, A. (2009). *Discovering Statistics Using SPSS (3rd edn).* London: SAGE.

Filion, D. L., Dawson, M. E., Schell, A. M., & Hazlett, E. A. (1991). The relationship between skin conductance orienting and the allocation of processing. *Psychophysiology*, *28*, 410–424.

Foa, E. B., Dancu, C. V., Hembree, E. A., Jaycox, L. H., Meadows, E. A., & Street, G. (1999). A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *Journal of Consulting and Clinical Psychology*, *67*(2), 194–200.

Foa, E. B., Franklin, M. E., & Moser, J. (2002). Context in the clinic: how well do cognitive-behavioral therapies and medications work in combination? *Biological Psychiatry*, *52*, 987–97.

Foa, E. B., & Kozak, M. J. (1986). Emotional Processing of Fear: Exposure to Corrective Information. *Psychological Bulletin*, *99*(1), 20–35.

Foa, E. B., Molnar, C., & Cashman, L. (1995). Change in Rape Narratives During Exposure Therapy for Posttraumatic Stress Disorder. *Journal of Traumatic Stress*, *8*(4).

Foa, E. B., Riggs, D. S., Massie, E. D., & Yarczower, M. (1995). The Impact of Fear Activation and Anger on the Efficacy of Exposure Treatment for Posttraumatic Stress Disorder. *Behavior Therapy*, *26*, 487–499.

Foa, E. B., & Rothbaum, B. O. (2001). *Treating the trauma of rape: Cognitive-behavioral therapy for PTSD.*The Guilford Press.

Foa, E. B., Rothbaum, B. O., Riggs, D. S., & Murdock, T. B. (1991). Treatment of posttraumatic stress disorder in rape victims: a comparison between cognitive-behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology*, *59*(5), 715–23.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy*, *20*(2), 155–176.

Foa, E. B., Zinbarg, R. E., & Rothbaum, B. O. (1992). Uncontrollability and Unpredicatbility in Post-Traumatic Stress Disorder: An Animal Model. *Psychological Bulletin*, *112*(2), 218–238.

Foa, E. B., Zoellner, L. A., & Feeny, N. C. (2006). An Evaluation of Three Brief Programs for Facilitating Recovery After Assault. *Journal of Traumatic Stress*, *19*(1), 29–43.

Foa, E. B., Zoellner, L. A., Feeny, N. C., Hembree, E. a., & Alvarez-Conrad, J. (2002). Does imaginal exposure exacerbate PTSD symptoms? *Journal of Consulting and Clinical Psychology*, *70*(4), 1022–1028.

Foa, Edna B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress*, *6*(4), 459–473.

Genest, M., Levine, J., Ramsden, V., & Swanson, R. (1990). The impact of providing help: Emergency workers and cardiopulmonary resuscitation attempts. *Journal of Traumatic Stress*, *3*(2), 305–313.

Gillespie, K., Duffy, M., Hackmann, A., & Clark, D. M. (2002). Community based cognitive therapy in the treatment of post- traumatic stress disorder following the Omagh bomb. *Behaviour Research and Therapy*, *40*, 345–357.

Greenberg, L. S., & Pascual-leone, A. (2006). Emotion in Psychotherapy: A Practice-Friendly Research Review, *62*(5), 611–630.

Greenberg, P. E., Sistsky, T., Kessler, R. C., Finkelstein, S. N., Berndt, E. R., Davidson, J. R. T., Ballenger, J. C., et al. (1999). The Economic Burden of Anxiety Disorders in the 1990s. *Journal of Clinical Psychiatry*, *60*, 437–435.

Guthrie, R. M., & Bryant, R. A. (2006). Extinction learning before trauma and subsequent posttraumatic stress. *Psychosomatic Medicine*, *68*(2), 307–11.

Hackmann, A., Clark, D. M., & McManus, F. (2000). Recurrent images and early memories in social phobia. *Behaviour Research and Therapy*, *38*(6), 601–10.

Hackmann, A., Ehlers, A., Speckens, A., & Clark, D. M. (2004). Characteristics and Content of Intrusive Memories in PTSD and Their Changes With Treatment. *Journal of Traumatic Stress*, *17*(3), 231–240.

Hagenaars, M. A., & Arntz, A. (2012). Reduced intrusion development after post-trauma imagery rescripting; an experimental study. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(2), 808–14.

Hagenaars, M. A., Minnen, A. Van, A, E., Brewin, C. R., & Hoogduin, K. A. L. (2008). The effect of hypnotically induced somatoform dissociation on the development of intrusions after an aversive film. *Cognition & Emotion*, *22*(5), 944–963.

Halligan, S. L., Clark, D. M., & Ehlers, A. (2002). Cognitive processing, memory, and the development of PTSD symptoms: two experimental analogue studies. *Journal of Behavior Therapy and Experimental Psychiatry*, *33*(2), 73–89.

Halligan, S. L., Michael, T., Clark, D. M., & Ehlers, A. (2003). Posttraumatic stress disorder following assault: The role of cognitive processing, trauma memory, and appraisals. *Journal of Consulting and Clinical Psychology*, *71*(3), 419–431.

Hamm, A. O., & Vaitl, D. (1996). Affective learning: awareness and aversion. *Psychophysiology*, *33*, 698–710.

Hartley, C. A., & Phelps, E. A. (2010). Changing Fear: The Neurocircuitry of Emotion Regulation. *Neuropsychopharmacology*, *35*, 136–146.

Harvey, A. G., & Bryant, R. A. (1998). The relationship between acute stress disorder and posttraumatic stress disorder: a prospective evaluation of motor vehicle accident survivors. *Journal of Consulting and Clinical Psychology*, *66*(3), 507–12.

Harvey, A. G., & Bryant, R. A. (1999). A qualitative investigation of the organization of traumatic memories. *British Journal of Clinical Psychology*, *38*, 401–405.

Hidalgo, R. B., & Davidson, J. R. T. (2000). Posttraumatic Stress Disorder: Epidemiology and Health-Related Considerations. *Journal of Clinical Psychiatry*, *61*, 5–13.

Hirsch, C. R., Clark, D. M., Mathews, A., & Williams, R. (2003). Self-images play a causal role in social phobia. *Behaviour Research and Therapy*, *41*(8), 909–921.

Hofmann, S. G. (2009). Cognitive processes during fear acquisition and extinction in animals and humans: Implications for exposure therapy of anxiety disorders. *Clinical Psychology Review*, *28*(2), 199–210.

Holmes, E. A., Arntz, A., & Smucker, M. R. (2007). Imagery rescripting in cognitive behaviour therapy: images, treatment techniques and outcomes. *Journal of Behavior Therapy and Experimental Psychiatry*, *38*(4), 297–305.

Holmes, E. A., & Bourne, C. (2008). Inducing and modulating intrusive emotional memories: A review of the trauma film paradigm. *Acta Psychologica*, *127*, 553–566.

Holmes, E. A., Brewin, C. R., & Hennessy, R. G. (2004). Trauma Films, Information Processing, and Intrusive Memory Development. *Journal of Experimental Psychology*, *133*(1), 3–22.

Holmes, E. A., Grey, N., & Young, K. A. D. (2005). Intrusive images and "' hotspots '" of trauma memories in Posttraumatic Stress Disorder: an exploratory investigation of emotions and cognitive themes. *Journal of Behavior Therapy and Experimental Psychiatry*, *36*, 3–17.

Holmes, E. A., James, E. L., Coode-Bate, T., & Deeprose, C. (2009). Can Playing the Computer Game "' Tetris '" Reduce the Build-Up of Flashbacks for Trauma? A Proposal from Cognitive Science. *PLoS ONE*, *4*(1), e4153.

Horowitz, M. J. (1975). Intrusive and repetitive thoughts after experimental stress. A summary. *Archives of General Psychiatry*, *32*, 1457–63.

Horowitz, M., Wilner, N., & Alvarez, W. (1979). Impact of Event Scale: a measure of subjective stress. *Psychosomatic Medicine*, *41*(3), 209–18.

Hunt, M. G. (1998). The only way out is through: emotional processing and recovery after a depressing life event. *Behaviour research and therapy*, *36*(4), 361–84.

Hunt, M. G., & Fenton, M. (2007). Imagery rescripting versus in vivo exposure in the treatment of snake fear. *Journal of Behavior Therapy and Experimental Psychiatry*, *38*(4), 329–44.

Izard, C. E. (1992). Basic emotions, relations among emotions, and emotion-cognition relations. *Psychological review*, *99*(3), 561–5.

Jaycox, L. H., Foa, E. B., & Morral, a R. (1998). Influence of emotional engagement and habituation on exposure therapy for PTSD. *Journal of Consulting and Clinical Psychology*, *66*(1), 185–92.

Jones, J., & Barlow, D. (1990). The Etiology of Post-traumatic Stress Disorder. *Clinical Psychology Review*, *10*, 299–328.

Joseph, S., & Williams, R. (1997). Attitudes towards emotional expression and post-traumatic stress in survivors of the Herald of Free Enterprise disaster. *British Journal of Clinical Psychology*, *36*, 133–138.

Jovanovic, T., Phifer, J. E., Sicking, K., Weiss, T., Norrholm, S. D., Bradley, B., & Ressler, K. J. (2011). Cortisol suppression by dexamethasone reduces exaggerated fear responses in posttraumatic stress disorder. *Psychoneuroendocrinology*, *36*(10), 1540–1552.

Keane, T. M., Kolb, L. C., Kaloupek, D. G., Orr, S. P., Blanchard, E. B., Thomas, R. G., Hsieh, F. Y., et al. (1998). Utility of psychophysiological measurement in the diagnosis of posttraumatic stress disorder: results from a Department of Veterans Affairs Cooperative Study. *Journal of Consulting and Clinical Psychology*, *66*(6), 914–23.

Kearns, M. C., Ressler, K. J., Zatzick, D., & Rothbaum, B. O. (2012). Early interventions for PTSD: a review. *Depression and Anxiety*, *842*, 833–842.

Kessler, R. C., Sonnega, A., Nelson, C. B., & Bromet, E. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, *52*(12), 1048–1060.

Kindt, M., & Soeter, M. (2011). Reconsolidation in a human fear conditioning study: a test of extinction as updating mechanism. *Biological Psychology*, *92*(1), 43–50.

Kindt, M., Van den Hout, M., & Buck, N. (2005). Dissociation related to subjective memory fragmentation and intrusions but not to objective memory disturbances. *Behaviour Therapy and Experimental Psychiatry*, *36*, 43–59.

Klucken, T., Kagerer, S., Schweckendiek, J., Tabbert, K., Vaitl, D., & Stark, R. (2009). Neural, electrodermal and behavioural response patterns in contingency aware and unaware subjects during a picture-picture conditioning paradigm. *Neuroscience*, *158*(2), 721–731.

Koenen, K. C., Nugent, N. R., & Amstadter, A. B. (2008). Gene-environment interaction in posttraumatic stress disorder: Review, strategy and new directions for future research. *Clinical Neuroscience*, *258*, 82–96.

Koren, D., Arnon, I., Lavie, P., & Klein, E. (2002). Sleep complaints as early predictors of posttraumatic stress disorder: a 1-year prospective study of injured survivors of motor vehicle accidents. *The American Journal of Psychiatry*, *159*(5), 855–7.

Koster, E. H. W., Verschuere, B., Crombez, G., & Van Damme, S. (2005). Time-course of attention for threatening pictures in high and low trait anxiety. *Behaviour Research and Therapy*, *43*(8), 1087–98.

Kozak, M. J., Foa, E. B., & Steketee, G. (1988). Process and Outcome of Exposure Treatment with Obsessive-Compulsives: Psychophysiological Indicators of Emotional Processing. *Behavior Therapy*, *19*, 157–169.

Krakow, B., Johnston, L., Melendrez, D., Hollifield, M., Warner, T. D., Chavez-Kennedy, D., & Herlan, M. J. (2001). Cognitive Behavior Therapy for Nightmares and Insomnia in Crime Victims With PTSD. *American Journal of Psychitary*, *158*, 2043–2047.

Krans, J., Naring, G., & Becker, E. S. (2009). Count out your intrusions: effects of verbal encoding on intrusive memories. *Memory*, *17*(8), 809–15.

Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2001). The PHQ-9. *Journal of General Internal Medicine*, *16*, 606–613.

Kroenke, K., Spitzer, R. L., Williams, J., Monahan, P. O., & Lowe, B. (2007). Anxiety disorders in primary care: prevalence, impairment, comorbidity, and detection. *Annals of Internal Medicine*, *146*, 317–326.

Kulka, R., Schlenger, W., Fairbank, J., Hough, R., Jordan, B., Marmar, C., & Weiss, D. S. (1990). *Trauma and the Vietnam War Generation.* New York, NY: Brunner/ Mazel.

Kupfer, D. J., Kuhl, E. A., & Regier, D. A. (2013). DSM-5-The Future Arrived. *Journal of the American Medical Association*, 1–2.

Kuyken, W., & Brewin, C. R. (1994). Intrusive memories of childhood abuse during depressive episodes. *Behaviour Research and Therapy*, *32*(5), 525–8.

Lang, P. J. (1985). The cognitive psychophysiology of emotion: Fear and anxiety. In Tuma, A. H.; Maser, J. D. (Eds). *Anxiety and the anxiety disorders.*, (pp 131-170), Hillsdale, NJ, England: Lawrence Erlbaum Associate.

Laposa, J. M., & Alden, L. E. (2006). An analogue study of intrusions. *Behaviour Research and Therapy*, *44*(7), 925–46.

Laposa, J. M., & Rector, N. A. (2012). The prediction of intrusions following an analogue traumatic event: peritraumatic cognitive processes and anxiety-focused rumination versus rumination in response to intrusions. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(3), 877–83.

Lawrence, J. W., Fauerbach, J., & Munster, A. (1996). Early avoidance of traumatic stimuli predicts chronicity of intrusive thoughts following burn injury. *Behaviour Research and Therapy*, *34*(8), 643–646.

Lazarus, R. S., Opton, E. M., Nomikos, M., & Rankin, N. (1965). The principle of short-circuiting of threat: further evidence. *Journal of Personality*, *33*(4), 622–635.

Leary, M. R., Adams, C. E., & Tate, E. B. (2006). Hypo-egoic self-regulation: exercising self-control by diminishing the influence of the self. *Journal of Personality*, *74*(6), 1803–31.

Lee, J. L. C., Milton, A. L., & Everitt, B. J. (2006). Reconsolidation and Extinction of Conditioned Fear: Inhibition and Potentiation. *Clinical Neuroscience*, *26*, 10051–10056.

Lindauer, R. T. L., Van Meijel, E. P. M., Jalink, M., Olff, M., Carlier, I. V. E., & Gersons, B. P. R. (2006). Heart rate responsivity to script-driven imagery in posttraumatic stress disorder: specificity of response and effects of psychotherapy. *Psychosomatic Medicine*, *68*, 33–40.

Lissek, S., Powers, A. S., McClure, E. B., Phelps, E. A., Woldehawariat, G., Grillon, C., & Pine, D. S. (2005). Classical fear conditioning in the anxiety disorders: a meta-analysis. *Behaviour Research and Therapy*, *43*, 1391–424.

Lommen, M. J. J., Engelhard, I. M., Sijbrandij, M., Van den Hout, M. A., & Hermans, D. (2013). Pre-trauma individual differences in extinction learning predict posttraumatic stress. *Behaviour Research and Therapy*, *51*(2), 63–67.

Lovibond, P. F., Davis, N. R., & O'Flaherty, A. S. (2000). Protection from extinction in human fear conditioning. *Behaviour Research and Therapy*, *38*(10), 967–83.

Mace, J. H. (2005). Priming involuntary autobiographical memories. *Memory*, *13*(8), 874–84.

Macklin, L., Metzger, L. J., Lasko, N. B., Orr, S. P., Pitman, R. K., & Berry, J. (2000). Five-year follow-up study of eye movement desensitization and reprocessing therapy for combat related posttraumatic stress disorder. *Comprehensive Psychiatry*, *41*(1), 24–27.

Magruder, K. M., Frueh, B. C., Knapp, R. G., Johnson, M. R., Vaughan, J. A., Carson, T. C., Powell, D. A., et al. (2004). PTSD symptoms, demographic characteristics, and functional status among veterans treated in VA primary care clinics. *Journal of Traumatic Stress*, *17*(4), 293–301.

Marks, I., Lovell, K., Noshirvani, H., Livanou, M., & Thrasher, S. (1998). Treatment of Posttraumatic Stress Disorder by Exposure and/or Cognitive Restructuring: A Controlled Study. *Archives of General Psychiatry*, *55*(4), 317–325.

Marx, B. P., Doron-Lamarca, S., Proctor, S. P., & Vasterling, J. J. (2009). The influence of pre-deployment neurocognitive functioning on post-deployment PTSD symptom outcomes among Iraq-deployed Army soldiers. *Journal of the International Neuropsychological Society*, *15*, 840–52.

Mayou, R. A., Ehlers, A., & Bryant, B. (2002). Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study. *Behaviour Research and Therapy*, *40*(6), 665–75.

Mayou, R. A., Ehlers, A., & Hobbs, M. (2000). Psychological debriefing for road traffic accident victims: Three-year follow-up of a randomised controlled trial. *British Journal of Psychiatry*, *176*, 589–593.

McFarlane, A. C., Barton, C. A., Yehuda, R., & Wittert, G. (2011). Cortisol response to acute trauma and risk of posttraumatic stress disorder. *Psychoneuroendocrinology*, *36*(5), 720–7.

Mcgaugh, J. L. (2000). Memory — a Century of Consolidation. *Science*, *287*, 248–251.

Mechanic, M. B., Resick, P. A., & Griffin, M. G. (1998). A comparison of normal forgetting, psychopathology, and information-processing models of reported amnesia for recent sexual trauma. *Journal of Consulting and Clinical Psychology*, *66*(6), 948–57.

Michael, T., Ehlers, A., Halligan, S. L., & Clark, D. M. (2005). Unwanted memories of assault: what intrusion characteristics are associated with PTSD? *Behaviour Research and Therapy*, *43*, 613–628.

Milad, M. R., Quinn, B. T., Pitman, R. K., Orr, S. P., Fischl, B., & Rauch, S. L. (2005). Thickness of ventromedial prefrontal cortex in humans is correlated with extinction memory. *Proceedings of the National Academy of Sciences of the United States of America*, *102*(30), 10706–10711.

Miller, M. W., Mckinney, A. E., Kanter, F. S., Korte, K. J., & Lovallo, W. R. (2011). Hydrocortisone suppression of the fear-potentiated startle response and posttraumatic stress disorder. *Psychoneuroendocrinology*, *36*(7), 970–980.

Mineka, S., & Oehlberg, K. (2008). The relevance of recent developments in classical conditioning to understanding the etiology and maintenance of anxiety disorders. *Acta Psychologica*, *127*(3), 567–80.

Mineka, S., & Öhman, A. (2002). Phobias and Preparedness : The Selective, Automatic, and Encapsulated Nature of Fear. *Biological Psychiatry*, *52*, 927–937.

Missirlian, T. M., Toukmanian, S. G., Warwar, S. H., & Greenberg, L. S. (2005). Emotional arousal, client perceptual processing, and the working alliance in experiential psychotherapy for depression. *Journal of Consulting and Clinical Psychology*, *73*(5), 861–71.

Monfils, M.-H., Cowansage, K. K., Klann, E., & LeDoux, J. E. (2009). Extinction-reconsolidation boundaries: key to persistent attenuation of fear memories. *Science*, *324*, 951–5.

Morrison, A. P., Beck, A. T., Glentworth, D., Dunn, H., Reid, G. S., Larkin, W., & Williams, S. (2002). Imagery and psychotic symptoms: a preliminary investigation. *Behaviour Research and Therapy*, *40*(9), 1053–62.

Mowrer, O. H. (1960). *Learning theory and behavior.* New York: Wiley.

Murray, J., Ehlers, A., & Mayou, R. (2002). Dissociation and post-traumatic stress disorder: two prospective studies of road traffic accident survivors. *The British Journal of Psychiatry*, *180*(4), 363–368.

Myers, K. M., Ressler, K. J., & Davis, M. (2006). Different mechanisms of fear extinction dependent on length of time since fear acquisition. *Learning & Memory*, *13*, 216–223.

Nader, K., Schafe, G. E., & Le Doux, J. E. (2000). Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. *Nature*, *406*, 722–726.

National Institute of Clinical Excellence. (2005). *CG26 Post-traumatic stress disorder (PTSD): full guideline, including appendices 1-13.* London: The Stationary Office, (26).

Nemeroff, C. B., Bremner, J. D., Foa, E. B., Mayberg, H. S., North, C. S., & Stein, M. B. (2006). Posttraumatic stress disorder: A state-of-the-science review. *Journal of Psychitric Research*, *40*, 1–21.

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: the 1989 Loma Prieta Earthquake. *Journal of Personality and Social Psychology*, *61*(1), 115–21.

Norrholm, S. D., Vervliet, B., Jovanovic, T., Boshoven, W., Myers, K. M., Davis, M., Rothbaum, B., et al. (2008). Timing of extinction relative to acquisition: a parametric analysis of fear extinction in humans. *Behavioral Neuroscience*, *122*(5), 1016–30.

O'Donnell, M. L., Elliott, P., Lau, W., & Creamer, M. (2007). PTSD symptom trajectories: from early to chronic response. *Behaviour Research and Therapy*, *45*(3), 601–6.

Öhman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: detecting the snake in the grass. *Journal of Experimental Psychology*, *130*, 466–478.

Orr, S. P., Lasko, N. B., Metzger, L. J., Berry, N. J., Ahern, C. E., & Pitman, R. K. (1998). Psychophysiologic assessment of women with posttraumatic stress disorder resulting from childhood sexual abuse. *Journal of Consulting and Clinical Psychology*, *66*(6), 906–13.

Orr, S. P., Metzger, L. J., Lasko, N. B., Macklin, M. L., Peri, T., & Pitman, R. K. (2000). De novo conditioning in trauma-exposed individuals with and without posttraumatic stress disorder. *Journal of Abnormal Psychology*, *109*(2), 290–298.

Orr, S. P., & Roth, W. T. (2000). Psychophysiological assessment: clinical applications for PTSD. *Journal of Affective Disorders*, *61*, 225–240.

Orr, S. P., Solomon, Z., Peri, T., Pitman, R. K., & Shalev, A. Y. (1997). Physiologic responses to loud tones in Israeli veterans of the 1973 Yom Kippur War. *Biological Psychiatry*, *41*(3), 319–26.

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2008). Predictors of Posttraumatic Stress Disorder and Symptoms in Adults: A Meta-Analysis. *Psychological Trauma: Theory, Research, Practice, and Policy*, *S*(1), 3–36.

Parslow, R. A., & Jorm, A. F. (2007). PTSD Symptoms in a Community Sample of Young Adults. *American Journal of Psychitary*, *164*(3), 509–515.

Paunovic, N., & Ost, L. G. (2001). Cognitive-behavior therapy vs exposure therapy in the treatment of PTSD in refugees. *Behaviour Research and Therapy*, *39*(10), 1183–97.

Pavlov, I. (1927). *Conditioned reflexes.* London: Oxford University Press.

Pearson, D. G., & Sawyer, T. (2011). Effects of Dual Task Interference on Memory Intrusions for Affective Images. *International Journal of Cognitive Therapy*, *4*(2), 122–133.

Peri, T., Ben-Shakhar, G., Orr, S. P., & Shalev, A. Y. (2000). Psychophysiologic Assessment of Aversive Conditioning in Posttraumatic Stress Disorder. *Biological Psychiatry*, *47*, 512–519.

Perkonigg, A., Kessler, R. C., Storz, S., & Wittchen, H. U. (2000). Traumatic events and post-traumatic stress disorder in the community: prevalence, risk factors and comorbidity. *Acta Psychiatrica Scandinavica*, *101*, 46–59.

Pineles, S. L., Orr, M. R., & Orr, S. P. (2009). An alternative scoring method for skin conductance responding in a differential fear conditioning paradigm with a long-duration conditioned stimulus. *Psychophysiology*, *46*, 984–995.

Pitman, R. K. (1989). Post-traumatic stress disorder, hormones, and memory. *Biological Psychiatry*, *26*(3), 221–3.

Pitman, R. K., Orr, S. P., Altman, B., Longpre, R. E., Poiré, R. E., Macklin, M. L., Michaels, M. J., et al. (1996). Emotional processing and outcome of imaginal flooding therapy in Vietnam veterans with chronic posttraumatic stress disorder. *Comprehensive Psychiatry*, *37*(6), 409–18.

Pitman, R. K., Orr, S. P., Forgue, D. F., De Jong, J. B., & Claiborn, J. M. (1987). Psychophysiologic assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. *Archives of general psychiatry*, *44*(11), 970–5.

Rachman, S., & Lopatka, C. (1988). Return of fear: underlearning and overlearning. *Behaviour Research and Therapy*, *26*, 99–104.

Rauch, S. L., Shin, L. M., & Phelps, E. A. (2006). Neurocircuitry models of posttraumatic stress disorder and extinction: human neuroimaging research-past, present, and future. *Biological Psychiatry*, *60*(4), 376–82.

Rescorla, R. A. (1973). Effect of US habituation following conditioning. *Journal of Comparative Physiology and Psychology*, *82*, 137—143.

Resnick, H., Acierno, R., Holmes, M., Kilpatrick, D. G., & Jager, N. (1999). Prevention of Post-Rape Psychopathology: Preliminary Findings of a Controlled Acute Rape Treatment Study. *Journal of Anxiety Disorders*, *13*(4), 359–370.

Richard, D. C., & Lauterbach, D. (2007). *Handbook of exposure therapies.* Academic Press.

Richards, D. A., & Rose, J. S. (1991). Exposure therapy for post-traumatic stress disorder. Four case studies. *The British Journal of Psychiatry*, *158*(6), 836–840.

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: the cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology*, *79*(3), 410–24.

Rose, S. C., Bisson, J., Churchill, R., & Wessely, S. (2002). Psychological debriefing for preventing post traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews*, (2).

Rothbaum, B. O., & Davis, M. (2003). Applying Leaning Principles to the Treatment of Post-Trauma Reactions. *Annals of the New York Academy of Sciences*, *1008*(1), 112–121.

Rothbaum, B. O., Foa, E. B., Riggs, D. S., Murdock, T., & Walsh, W. (1992). A prospective examination of post-traumatic stress disorder in rape victims. *Journal of Traumatic Stress*, *5*(3), 455–475.

Rothbaum, B. O., Houry, D., Heekin, M., Leiner, A. S., Daugherty, J., Smith, L. S., & Gerardi, M. (2008). A pilot study of an exposure-based intervention in the ED designed to prevent posttraumatic stress disorder. *The American Journal of Emergency Medicine*, *26*, 326–330.

Rothbaum, B. O., Kearns, M. C., Price, M., Malcoun, E., Davis, M., Ressler, K. J., Lang, D., et al. (2012). Early intervention may prevent the development of posttraumatic stress disorder: a randomized pilot civilian study with modified prolonged exposure. *Biological Psychiatry*, *72*(11), 957–63.

Rothbaum, B. O., Kozak, M. J., Foa, E. B., & Whitaker, D. J. (2001). Posttraumatic Stress Disorder in Rape Victims: Autonomic Habituation to Auditory Stimuli. *Journal of Traumatic Stress*, *14*(2).

Sales, E., Baum, M., & Shore, B. (1984). Victim Readjustment Following Assault. *Journal of Social Issues*, *40*(1), 117–136.

Salmond, C. H., Meiser-Stedman, R., Glucksman, E., Thompson, P., Dalgleish, T., & Smith, P. (2010). The nature of trauma memories in acute stress disorder in children and adolescents. *Journal of Child Psychology and Psychiatry*, *52*(5), 560–70.

Schartau, P. E. S., Dalgleish, T., & Dunn, B. D. (2009). Seeing the bigger picture: training in perspective broadening reduces self-reported affect and psychophysiological response to distressing films and autobiographical memories. *Journal of Abnormal Psychology*, *118*(1), 15–27.

Schiller, D., Cain, C. K., Curley, N. G., Schwartz, J. S., Stern, S. A., Ledoux, J. E., & Phelps, E. A. (2008). Evidence for recovery of fear following immediate extinction in rats and humans. *Learning & Memory*, 394–402.

Schiller, D., Levy, I., Niv, Y., Ledoux, J. E., & Phelps, E. A. (2008). From Fear to Safety and Back: Reversal of Fear in the Human Brain. *Journal of Neuroscience*, *28*(45), 11517–11525.

Schiller, D., Monfils, M.-H., Raio, C. M., Johnson, D. C., Ledoux, J. E., & Phelps, E. A. (2010). Preventing the return of fear in humans using reconsolidation update mechanisms. *Nature*, *463*(7277), 49–53.

Scholes, C., Turpin, G., & Mason, S. (2007). A randomised controlled trial to assess the effectiveness of providing self-help information to people with symptoms of acute stress disorder following a traumatic injury. *Behaviour Research and Therapy*, *45*, 2527–2536.

Schwarz, E. D., Kowalski, J. M., & Mcnally, R. J. (1993). Malignant Memories: Post-lkaumatic Changes in Memory in Adults After a School Shooting. *Journal of Traumatic Stress*, *6*(4), 545–553.

Shalev, A. Y., Ankri, Y., Israeli-Shalev, Y., Peleg, T., Adessky, R., & Freedman, S. (2012). Prevention of posttraumatic stress disorder by early treatment: results from the Jerusalem Trauma Outreach And Prevention study. *Archives of General Psychiatry*, *69*(2), 166–76.

Shalev, A. Y., Freedman, S., Peri, T., Brandes, D., & Sahar, T. (1997). Predicting PTSD in trauma survivors: prospective evaluation of self-report and clinician-administered instruments. *The British Journal of Psychiatry*, *170*(6), 558–564.

Shalev, A. Y., & Rogel-Fuchs, Y. (1993). Psychophysiology of the posttraumatic stress disorder: from sulfur fumes to behavioral genetics. *Psychosomatic Medicine*, *55*(5), 413–23.

Shalev, A. Y., Sahar, T., Freedman, S., Peri, T., Glick, N., Brandes, D., Orr, S. P., et al. (1998). A prospective study of heart rate response following trauma and the subsequent development of posttraumatic stress disorder. *Archives of General Psychiatry*, *55*(6), 553–9.

Shapiro, F. (1989). Eye movement desensitization: a new treatment for post-traumatic stress disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *20*(3), 211–7.

Shore, J. H., Vollmer, W. M., & Tatum, E. L. (1989). Community Patterns of Posttraumatic Stress Disorders. *The Journal of Nervous and Mental Disease*, *177*, 681–685.

Sijbrandij, M., Olff, M., Reitsma, J. B., Carlier, I. V. E., & Gersons, B. P. R. (2006). Emotional or educational debriefing after psychological trauma: Randomised controlled trial. *British Journal of Psychiatry*, *189*, 150–155.

Silver, R. L., Boon, C., & Stones, M. H. (1983). Searching for Meaning in Misfortune: Making Sense of Incest. *Journal of Social Issues*, *39*(2), 81–101.

Smeets, G., Merckelbach, H., & Griez, E. (1997). Panic disorder and right-hemisphere reliance. *Anxiety, Stress & Coping*, *10*, 245–255.

Soeter, M., & Kindt, M. (2011). Disrupting reconsolidation: pharmacological and behavioral manipulations. *Learning & Memory*, *18*(6), 357–66.

Southwick, S. M., Morgan, A. C., Nicolaou, A. L., & Charney, D. S. (1997). Consistency of Memory for Combat-Related Traumatic Events in Veterans of Operation Desert Storm. *American Journal of Psychitary*, *152*(2), 173–177.

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P., & Jacobs, G. A. (1983). *Manual for the state-trait anxiety inventory.* Palo Alto, CA: Consulting Psychologists.

Spielberger, C. D., & Reheiser, E. C. (2003). Measuring anxiety, anger, depression, and curiosity as emotional states and personality traits with the STAI, STAXI, and STPI. In Hersen, M. (Ed). *Comprehensive handbook of psychological assessment, 2.* (pp. 70–86.). Wiley

Spitzer, R. L., Kroenke, K., & Williams, J. B. W. (1999). Validation and Utility of a Self-report Version of PRIME-MD: The PHQ Primary Care Study. *Journal of the American Medical Association*, *282*(18), 1737–1744.

Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder: the GAD-7. *Archives of Internal Medicine*, *166*(10), 1092–7.

Squire, L. R., & Davis, H. P. (1981). The Pharmacology of Memory: A Neuobiological Perspective. *Annual Review of Pharmacology and Toxicology*, *21*, 323–6.

Steel, C., Fowler, D., & Holmes, E. A. (2005). Trauma-Related Intrusions and Psychosis : An Information Processing Account. *Behavioural and Cognitive Psychotherapy*, *33*, 139–152.

Steil, R., & Ehlers, A. (2000). Dysfunctional meaning of posttraumatic intrusions in chronic PTSD. *Behaviour Research and Therapy*, *38*, 537–558.

Steil, R. I. (1997). *Posttraumatische intrusionen nach verkehrsunfällen. [Posttraumatic intrusions after road accidents].* Frankfurt, Germany: Peter Lang.

Stein, M. B., Walker, J. R., Hazen, A. L., & Forde, D. R. (1997). Full and partial posttraumatic stress disorder: findings from a community survey. *The American Journal of Psychiatry*, *154*(8), 1114–9.

Sterud, T., Ekeberg, Ø., & Hem, E. (2006). Health status in the ambulance services: a systematic review. *BMC health services research*, *6*(82).

Stuart, A. D. P., Holmes, E. A., & Brewin, C. R. (2006). The influence of a visuospatial grounding task on intrusive images of a traumatic film. *Behaviour Research and Therapy*, *44*(4), 611–9.

Sundin, E. C., & Horowitz, M. (2002). Impact of Event Scale: psychometric properties. *The British Journal of Psychiatry*, *180*(3), 205–209.

Tabbert, K., Stark, R., Kirsch, P., & Vaitl, D. (2006). Dissociation of neural responses and skin conductance reactions during fear conditioning with and without awareness of stimulus contingencies. *NeuroImage*, *32*, 761–770.

Tarrier, N., & Sommerfield, C. (2004). Treatment of Chronic PTSD by Cognitive Therapy and Exposure: 5-Year Follow-Up. *Behavior Therapy*, *35*, 231–246.

Tarrier, N., Sommerfield, C., Pilgrim, H., & Humphreys, L. (1999). Cognitive therapy or imaginal exposure in the treatment of post- traumatic stress disorder. Twelve-month follow-up. *The British Journal of Psychiatry*, *175*(6), 571–575.

Turpin, G., Downs, M., & Mason, S. (2005). Effectiveness of providing self-help information following acute traumatic injury: randomised controlled trial. *British Journal of Psychiatry*, *187*, 76–82.

Van Emmerik, A. A. P., Kamphuis, J. H., Hulsbosch, A. M., & Emmelkamp, P. M. G. (2002). Single session debriefing after psychological trauma: a meta-analysis. *Lancet*, *360*(9335), 766–71.

Vansteenwegen, D., Hermans, D., Vervliet, B., Francken, G., Beckers, T., Baeyens, F., & Eelen, P. (2005). Return of fear in a human differential conditioning paradigm caused by a return to the original acquistion context. *Behaviour Research and Therapy*, *43*, 323–336.

Vervliet, B., Vansteenwegen, D., & Eelen, P. (2004). Generalization of Extinguished Skin Conductance Responding in Human Fear Conditioning. *Learning & Memory*, *11*, 555–558.

Walker, M. P., Brakefield, T., & Hobson, J. A. (2003). Dissociable stages of human memory consolidation and reconsolidation. *Nature*, *425*, 616–620.

Watkins, E. (2004). Adaptive and maladaptive ruminative self-focus during emotional processing. *Behaviour research and therapy*, *42*(9), 1037–52.

Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, *3*, 1–14.

Weike, A. I., Schupp, H. T., & Hamm, A. O. (2007). Fear acquisition requires awareness in trace but not delay conditioning. *Psychophysiology*, *44*(1), 170–80.

Weiss, D. S., & Marmar, C. R. (1997). The Impact of Events Scale - Revised. In Wilson, P., & Keane, T.M. (Eds.). *Assessing Psychological Trauma and PTSD: A handbook for practitioners.*, (pp 399-411), New York: Guildford Press.

Weiss, D. S., Marmar, C. R., Metzler, T. J., & Ronfeldt, H. M. (1995). Predicting symptomatic distress in emergency services personnel. *Journal of Consulting and Clinical Psychology*, *63*(3), 361–8.

Wells, A., & Papageorgiou, C. (1995). Worry and the incubation of intrusive images following stress. *Behaviour Research and Therapy*, *33*(5), 579–583.

Whelton, W. J. (2004). Emotional processes in psychotherapy: evidence across therapeutic modalities. *Clinical Psychology & Psychotherapy*, *11*(1), 58–71.

White, R. (2012). The Effect of Processing Mode on Intrusive Memories and Emotional Reactivity following Exposure to Trauma: An Experimental Analogue Study. *Unpublished Doctorate in Clinical Psychology thesis, Institute of Psychiatry, King's College London.*

Wild, J., Hackmann, A., & Clark, D. M. (2007). When the present visits the past: updating traumatic memories in social phobia. *Journal of Behavior Therapy and Experimental Psychiatry*, *38*(4), 386–401.

Wilson, J. J. P., & Keane, T. M. (2004). *Assessing psychological trauma and PTSD. The Guilford Press.*

Woud, M. L., Holmes, E. A., Postma, P., Dalgleish, T., & Mackintosh, B. (2012). Ameliorating Intrusive Memories of Distressing Experiences Using Computerized Reappraisal Training. *Emotion*, *12*(4), 778 –784.

Yuan, C., Wang, Z., Inslicht, S. S., McCaslin, S. E., Metzler, T. J., Henn-Haase, C., Apfel, B. A., et al. (2011). Protective factors for posttraumatic stress disorder symptoms in a prospective study of police officers. *Psychiatry Research*, *188*(1), 45–50.

Zatzick, D., Roy-Byrne, P., Russo, J., Rivara, F., Droesch, R., Wagner, A., Dunn, C., et al. (2004). A Randomized Effectiveness Trial of Stepped Collaborative Care for Acutely Injured Trauma Survivors. *Archives of General Psychiatry*, *61*, 498–506.

Zetsche, U., Ehring, T., & Ehlers, A. (2009). The effects of rumination on mood and intrusive memories after exposure to traumatic material: An experimental study. *Journal of Behavior Therapy and Experimental Psychiatry*, *40*(4), 499–514.

Zinbarg, R. E., & Mohlman, J. (1998). Individual Differences in the Acquisition of Affectively Valenced Associations. *Journal of Personality and Social Psychology*, *74*(4), 1024–1040.

Zohar, J., Yahalom, H., Kozlovsky, N., Cwikel-hamzany, S., Matar, M. A., Kaplan, Z., Yehuda, R., et al. (2011). High dose hydrocortisone immediately after trauma may alter the trajectory of PTSD: Interplay between clinical and animal studies. *European Neuropsychopharmacology*, *21*(11), 796–809.

6. Appendix

Appendix 1: Recruitment email

Circular email for use for recruitment of volunteers for study ref: PNM/11/12-35, approved by the Psychiatry, Nursing and Midwifery Research Ethics Sub-Committee (PNM RESC). This project contributes to the College's role in conducting research, and teaching research methods. You are under no obligation to reply to this email, however if you choose to, participation in this research is voluntary and you may withdraw at any time.

You are invited to take part in a research study which investigates ways to reduce the reoccurrence of unwanted thoughts, images and physiological reactions after a trauma. This study aims to understand why this happens so that methods may be developed for treating these reactions. In addition, you will receive £10 as compensation for your time.

We are recruiting people over the age of 18 who are fluent in English and not currently experiencing clinically significant mental health problems or a significant history of trauma.

If you agree to participate in this study, we will ask you to complete some questionnaires by email. These questionnaires will ask about symptoms of anxiety, depression and post-traumatic stress disorder and whether you have experienced any traumatic events. These questionnaires are for screening purposes only. We will then ask you to attend a session that lasts approximately 1 hour. In this session, you will be shown a series of short film clips that contain traumatic material (e.g. humans and animals in distress).Your skin conductance will be measured and you will be asked a few questions about the films. We will also ask you to complete a simple diary over the following week and some on-line questionnaires. These should take no more than 15 minutes to complete. Sessions will take place at the Institute of Psychiatry in Denmark Hill.

Your participation is entirely voluntary and if you decide to take part but later wish to withdraw, you may do so at any time without giving a reason. All information will be anonymised and kept confidential.

If you are interested in taking part in this study or would like more information then please email victoria.pile@kcl.ac.uk. You will be given an information sheet with further details.

Kind regards,

Victoria Pile
Trainee Clinical Psychologist
Institute of Psychiatry, Kings College London
Department of Psychology|3rd Floor Addiction Sciences Building|4 Windsor Walk|
Denmark Hill|London|SE5 8AF
Telephone: 020 7848 0733 Email: victoria.pile@kcl.ac.uk

Appendix 2: Letter of ethical approval

Victoria Pile
Department of Psychology
Institute of Psychiatry
Addiction Sciences Building
4 Windsor Walk
London SE5 8AF

28 March 2012

Dear Victoria

PNM/11/12-35 Investigating factors linked to trauma reactions and recovery.

Review Outcome: Full Approval

Thank you for sending in the amendments/clarifications requested to the above project. I am pleased to inform you that these meet the requirements of the PNM RESC and therefore that full approval is now granted with the following proviso:

1. Information Sheets: Explicitly state that potential participants who are screened and then identified as having clinically significant levels of anxiety, depression, current PTSD symptoms (explain in lay terms what this is) or a history of trauma will be excluded from the study.

Please ensure that you follow all relevant guidance as laid out in the King's College London Guidelines on Good Practice in Academic Research (http://www.kcl.ac.uk/college/policyzone/index.php?id=247).

For your information ethical approval is granted until **28 March 2015**. If you need approval beyond this point you will need to apply for an extension to approval at least two weeks prior to this explaining why the extension is needed, (please note however that a full re-application will not be necessary unless the protocol has changed). You should also note that if your approval is for one year, you will not be sent a reminder when it is due to lapse.

Ethical approval is required to cover the duration of the research study, up to the conclusion of the research. The conclusion of the research is defined as the final date or event detailed in the study description section of your approved application form (usually the end of data collection when all work with human participants will have been completed), not the completion of data analysis or publication of the results. For projects that only involve the further analysis of pre-existing data, approval must cover any period during which the researcher will be accessing or evaluating individual sensitive and/or un-anonymised records. Note that after the point at which ethical approval for your study is no longer required due to the study being complete (as per the above definitions), you will still need to ensure all research data/records management and storage procedures agreed to as part of your application are adhered to and carried out accordingly.

If you do not start the project within three months of this letter please contact the Research Ethics Office.

Should you wish to make a modification to the project or request an extension to approval you will need approval for this and should follow the guidance relating to modifying approved applications: http://www.kcl.ac.uk/innovation/research/support/ethics/applications/modifications.aspx
The circumstances where modification requests are required include the addition/removal of participant groups, additions/removal/changes to research methods, asking for additional data from participants, extensions to the ethical approval period. Any proposed modifications should only be carried out once full approval for the modification request has been granted.

Any unforeseen ethical problems arising during the course of the project should be reported to the

approving committee/panel. In the event of an untoward event or an adverse reaction a full report must be made to the Chair of the approving committee/review panel within one week of the incident.

Please would you also note that we may, for the purposes of audit, contact you from time to time to ascertain the status of your research.

If you have any query about any aspect of this ethical approval, please contact your panel/committee administrator in the first instance (http://www.kcl.ac.uk/innovation/research/support/ethics/contact.aspx). We wish you every success with this work.

With best wishes

Yours sincerely

Catherine Fieulleteau
Senior Research Ethics Officer

Cc: Dr Jennifer Wild

Appendix 3: Letter of ethical approval for modifications

Victoria Pile
Department of Psychology
Institute of Psychiatry
Addiction Sciences Building
4 Windsor Walk
London SE5 8AF

10 September 2012

Dear Victoria

PNM/11/12-35 Investigating factors linked to trauma reactions and recovery.

Thank you for submitting a modification request for the above study. I am writing to confirm approval of this. The modifications are summarised below:

1. Participants will attend one session, rather than two.
2. Participants will receive £10 compensation for their time.

If you have any questions regarding this application please contact the Research Ethics Office.

Yours sincerely

Catherine Fieulleteau
Senior Research Ethics Officer

Appendix 4: General Information Questionnaire

General Information Questionnaire

Please give us some background information about yourself.
The following questions ask about you and your life in general. For each question, either write the answer on the line or tick the box which most applies to you. Some questions may have more than one answer.

1. Date of birth	_____/_____/_____
2. Gender	❑ male ❑ female
3. Ethnic background	❑ Caucasian (White) ❑ Black (Caribbean, African, Other) ❑ Pacific Asian ❑ Indian/Pakistani/Bangladeshi ❑ Other _____
4. Is English your first language?	❑ Yes ❑ No. Which is your first language? _____
5. What is your marital status?	❑ single ❑ divorced/separated ❑ married ❑ widowed
6. Are you	❑ employed full-time ❑ a full-time student ❑ employed part-time ❑ a part-time student ❑ self-employed ❑ unemployed ❑ a homemaker ❑ on disability ❑ on sick leave ❑ retired ❑ other: _____
7. What is your job/ course? (If unemployed/ retired: What was your last job?)	_____
8. At what age did you start/finish education?	start: _____ finish: _____
9. Please mark any qualifications you have.	❑ No exams _____ ❑ Degree ❑ Other: ❑ GCSE/O Levels/GNVQ ❑ Postgraduate degree: please circle: Masters / PhD / other:_____ ❑ A Levels/NVQ ❑ Vocational degree
10. Please try to estimate approximately your annual household income.	❑ Less than £5000 per year ❑ £20,000 to £30,000 per year ❑ £5000 to £10,000 per year ❑ £30,000 to £40,000 per year ❑ £10,000 to 15,000 per year ❑ £40,000 to £50,000 per year ❑ £15,000 to £20,000 per year ❑ over £50,000 per year

Appendix 5: Trauma screener

Many people have lived through or witnessed a very stressful and traumatic event at some point in their lives. Indicate whether or not you have experienced each traumatic event listed below by marking **Y** for Yes or **N** for No.

				If YES, did you experience fear, helplessness or horror as a result of the event?		If YES, did this incident happen to you in childhood (C) or adulthood (A)?	
1.	Serious traffic accident, (e.g., car, bike, train, or boating accident)	Y	N	Y	N	C	A
2.	Serious other accident, fire, or explosion (for example, accident at work, fire at home)	Y	N	Y	N	C	A
3.	Natural disaster (for example, tornado, hurricane, flood, or major earthquake)	Y	N	Y	N	C	A
4.	Non-sexual assault (for example, being mugged, physically attacked, shot, stabbed, or held at gunpoint)	Y	N	Y	N	C	A
5.	Seriously injuring or killing someone else	Y	N	Y	N	C	A
6.	Sexual assault (for example, rape or attempted rape)	Y	N	Y	N	C	A
7.	Military combat or a war zone	Y	N	Y	N	C	A
8.	Terrorist attack (e.g., bombing)	Y	N	Y	N	C	A
9.	Unwanted sexual contact when you were younger than 18 with someone who was 5 or more years older than you (for example, contact with genitals, breasts)	Y	N	Y	N	C	A
10.	Imprisonment (for example, prisoner of war, hostage)	Y	N	Y	N	C	A
11.	Torture	Y	N	Y	N	C	A
12.	Life-threatening illness	Y	N	Y	N	C	A
13.	Witnessing others die / being seriously hurt	Y	N	Y	N	C	A
14.	Sudden, traumatic death of significant other	Y	N	Y	N	C	A
15.	Life-threatening illness of significant other	Y	N	Y	N	C	A
16.	Serious risk of contamination by another person	Y	N	Y	N	C	A
17.	Cot death	Y	N	Y	N	C	A
18.	Witnessed or come across a suicide	Y	N	Y	N	C	A
19.	Threatened or harassed by someone without a weapon	Y	N	Y	N	C	A
20.	Serious ongoing physical or emotional abuse as a child	Y	N	Y	N	C	A
21.	Suffered a great shock because one of the events on the list happened to someone close to you	Y	N	Y	N	C	A
22.	Other traumatic event	Y	N	Y	N	C	A

If other, please specify……

Please select from the events that you have experienced which one you would consider to have been your most stressful life event……

When did this event take place (approximate date)? ……

Appendix 6: Subjective ratings of distress

Subjective ratings of distress

Participant Code:
Date
Phase:

---

1. How distressed does the red square make you feel?

10 20 30 40 50 60 70 80 90 100

Not at all distressed extremely distressed

2. How distressed does the blue circle make you feel?

10 20 30 40 50 60 70 80 90 100

Not at all distressed extremely distressed

Appendix 7: Intrusions diary

Diary of intrusions

In this diary, please note every time you have a spontaneously occurring memory (an intrusive image or thought which is not deliberately recalled) about any of the scenes that you saw in the films.

What time each day will you fill the diary in? _____

Days after watching the films	**Time of day**	**How many times have you had spontaneously occurring memories from the films?**	**What was the memory?** Was it a image (I) or a thought (T)	**How distressing did you find this memory?** Rating it from 0 (not distressing at all)to 10 (extremely distressing)
DAY 1	Morning			
	Afternoon			
	Evening			
DAY 2	Morning			
	Afternoon			
	Evening			
DAY 3	Morning			
	Afternoon			
	Evening			
DAY 4	Morning			
	Afternoon			

	Evening			
DAY 5	Morning			
	Afternoon			
	Evening			
DAY 6	Morning			
	Afternoon			
	Evening			
DAY 7	Morning			
	Afternoon			
	Evening			

Appendix 8: Diary compliance

Diary Compliance

Participant Code:
Date:

How accurate do you think your completion of the diary has been?

1 2 3 4 5 6 7 8 9 10

Not at all accurate **extremely accurate**

Please rate how true the following statement is:
"I have been unable (or have forgotten) to record my intrusive images in the diary"

1 2 3 4 5 6 7 8 9 10

Not at all true **extremely true of me**

Appendix 9: Response to Memories questionnaire

Response to memories of the trauma films

We are interested in how you responded when memories of the trauma film you watched last week, popped into your mind. There are many ways you might have dealt with such memories. Please circle the answer that applies best to you. There are no right or wrong answers.

What did you do when memories of the films popped into your mind?

1.	I tried to push them out of my mind.	Never	Sometimes	Often	Always
2.	I thought about how the people's lives in the films would never be the same again.	Never	Sometimes	Often	Always
3.	I detached myself from the memories.	Never	Sometimes	Often	Always
4.	I tried hard to control my emotions.	Never	Sometimes	Often	Always
5.	I reminded myself where the memory had come from.	Never	Sometimes	Often	Always
6.	I drifted off into a world of my own.	Never	Sometimes	Often	Always
7.	I kept thinking about why the distressing things had happened to the people in the films.	Never	Sometimes	Often	Always
8.	In my head, I carried the images/thoughts on past the most distressing bit.	Never	Sometimes	Often	Always
9.	I went onto automatic pilot and wasn't aware of what I was doing.	Never	Sometimes	Often	Always
10.	I drank alcohol, took medication or used drugs.	Never	Sometimes	Often	Always
11.	I went over what happened in the films again and again.	Never	Sometimes	Often	Always
12.	I thought about the positives of experiencing a negative event.	Never	Sometimes	Often	Always
13.	I felt disconnected from the memories and from myself.	Never	Sometimes	Often	Always
14.	I worried that something similar will happen to me or my family.	Never	Sometimes	Often	Always
15.	I thought of something else.	Never	Sometimes	Often	Always
16.	I did not respond in any way to the memories.	Never	Sometimes	Often	Always
17.	I distracted myself with something else.	Never	Sometimes	Often	Always
18.	I added a positive ending to the distressing parts of the memory.	Never	Sometimes	Often	Always
19.	I dwelt on what the people in the film should have done differently.	Never	Sometimes	Often	Always
20.	The memories didn't seem real to me, it just felt like I was watching someone else's experience.	Never	Sometimes	Often	Always
21.	I reminded myself that it was just a film that I had watched.	Never	Sometimes	Often	Always

Appendix 10: Pilot participant baseline measures

	Updating mean (SD) (n=7)	Exposure mean (SD) (n=3)	Neutral mean (SD) (n=3)	Exploratory analysis
Age	28.14 (2.67)	23.80 (1.15)	26 (3)	*H*(2)=1.22, p=.54
Sex	2 males, 5 females	3 females	3 females	$\chi^2(2)=2.03$, p =.36
IES-R	5.57 (7.37)	8.67 (15.01)	1.00 (1.00)	*H*(2)=0.050 p=.98
GAD-7	2.23 (2.21)	2.33 (1.53)	1.33 (1.15)	*H*(2)=0.62, p=.73
PHQ-9	2.14 (1.57)	2.67 (1.53)	1.00 (1.00)	*H*(2)=1.94, p=.38
STAI	32.43 (8.24)	43.67 (10.01)	26.67 (8.32)	*H*(2)=4.25, p=.12
Trauma screener	1.43 (1.27)	2.00 (1.00)	0.67 (1.15)	*H*(2)=2.00, p=.37

Pilot participants' outcome measures

	Updating mean (SD) (n=7)	Exposure mean (SD) (n=3)	Neutral mean (SD) (n=3)	Kruskal-Wallis
IES-R	5.00 (4.00)	10.33 (12.34)	10.00 (10.00)	*H*(2)=0.81, p=0.67
Intrusion frequency	4.57 (1.90)	5.33 (6.66)	7 (7.55)	*H*(2)=0.39, p=0.83
Intrusion distress rating	8.43 (8.48)	25 (33.95)	27.67 (24.80)	*H*(2)=0.88, p=0.65

Appendix 11: Information sheet and consent form

INFORMATION SHEET FOR PARTICIPANTS

REC Reference Number: PNM/11/12-35

YOU WILL BE GIVEN A COPY OF THIS INFORMATION SHEET

Investigating factors linked to trauma reactions and recovery

We would like to invite you to participate in this original research project that forms part of a doctoral thesis. You should only participate if you want to; choosing not to take part will not disadvantage you in any way. Before you decide whether you want to take part, it is important for you to understand why the research is being done and what your participation will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information.

Aims of the research
People who experience a traumatic event can develop post-traumatic stress disorder (PTSD). One of the main features of this disorder is to have images, thoughts and physiological reactions linked to the event that occur without warning, which can be is incredibly frightening for the individual. This study aims to understand why this happens so that methods may be developed for treating these reactions.

Who are we asking to take part?
We are recruiting people over the age of 18 who are fluent in English and not currently experiencing clinically significant mental health problems or a significant history of trauma.

What will happen if I agree to take part?
If you agree to participate in this study, we will ask you to complete some questionnaires that we will send to you and ask you to return by email. These questionnaires are for screening purposes only and will be destroyed immediately after use. These questionnaires will ask about symptoms of anxiety, depression and post-traumatic stress disorder and whether you have experienced any traumatic events. Symptoms of PTSD sometimes occur following a catastrophic or highly threatening event and include avoiding reminders of the trauma, having flashbacks and nightmares of the trauma and having poor concentration and feeling irritable. If the screening questionnaires indicate that you do have significant mental health problems then you will be thanked for your time and asked whether you would like to speak further with the researcher but excluded from the main part of the study. Data gathered from the screening stage, both from participants who enter the main study and those who do not, will be scored and the scores will be kept in an anonymised format.

The main study consists of one session and some follow-up questionnaires. The session lasts for about an hour and you will be shown a series of short film clips that contain traumatic material (e.g. humans in distress).Your skin conductance will be measured and you will be asked a few questions about the films. Skin conductance is a way of measuring the changes in electrical conductance of the skin. This is frequently used as a measure of psychological arousal. Skin conductance will be measured using a 4/30 Data acquisition system and by placing two electrodes on each of your wrists, these are harmless and do not hurt.

After the first session, you will be asked to complete a simple diary over the following week and then some questionnaires on-line one week later. The questionnaires should take no more than 15 minutes to complete. Sessions will take place at the Institute of Psychiatry in Denmark Hill.

Are there any risks in participating in this study?
The films that we are using do contain scenes of humans and animals in distress and so there is a risk that you may find these upsetting. However, any distress you may experience is likely to be short-lived and very similar studies have been conducted without any adverse consequences. In addition, the content of the clips will be similar to what you may see on a television program or news programs. If you do feel any distress during the session, then we would encourage you to speak to the researcher, Victoria Pile, who is a clinical psychologist in training. You will also be given her contact details to take away with you in case you feel distress in relation to the study later on. If you are still worried or distressed by the videos, then you are able to contact her supervisor, Dr Jennifer Wild who is a consultant clinical psychologist and who specialises in treating trauma reactions.

Your participation is entirely voluntary and so if you decide to take part but later wish to withdraw, you may do so at any time without giving a reason. All information will be anonymised and kept confidential.

Are there any benefits to taking part?
This study hopes to improve understanding of post-traumatic stress disorder and inform psychological interventions for it. In addition, you will receive £10 as compensation for your time. You will also be given the option of being sent a summary of the research findings.

Arrangements for ensuring confidentiality
To ensure the confidentiality, you will be randomly allocated a code. This unique code will be used on questionnaires, measures and data analysis so no personally identifiable information will be associated directly with your data. The consent form that you sign, if you choose to take part, will be kept separately from the data in a locked filing cabinet which only the main researcher has access to. After the study has been completed, any information linking you to your code will be destroyed. However, confidentiality will be broken in the unlikely event that you indicate potential harm to yourself or others as it is the researcher's duty to pass on this information. In this case, the researcher would inform her supervisor who would speak with you and possibly your GP if necessary.

It is up to you to decide whether to take part or not. If you decide to take part you are still free to withdraw at any time and without giving a reason. In addition to withdrawing yourself from the study, you may also withdraw any data/information you have already provided up until March 2013.

If this study has harmed you in any way you can contact King's College London using the details below for further advice and information:

Name and contact details of the researchers

Dr Jennifer Wild
Institute of Psychiatry (PO77), King's College London
De Crespigny Park
London
SE5 8AF
Telephone: 0207 848 5045 Email: jennifer.wild@kcl.ac.uk

Victoria Pile
Institute of Psychiatry, Kings College London
Department of Psychology, 3rd Floor Addiction Sciences Building
4 Windsor Walk, Denmark Hill, London
SE5 8AF
Telephone: 020 7848 0733 Email: victoria.pile@kcl.ac.uk

CONSENT FORM FOR PARTICIPANTS IN RESEARCH STUDIES

Investigating factors linked to trauma reactions and recovery

King's College Research Ethics Committee Ref: PNM/11/12-35

Thank you for considering taking part in this research.
Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research. If you have any questions about this project and what you are being invited to take part in, please ask the researcher before completing this form.

You will be given a copy of this Consent Form to keep and refer to at any time.

Please tick or initial

- **I understand that if I decide at any time during the research that I no longer wish to participate in this project, I can notify the researchers involved and withdraw from it immediately without giving any reason. Furthermore, I understand that I will be able to withdraw my data up March 2013.** ☐

- **I consent to the processing of my personal information for the purposes explained to me. I understand that such information will be handled in accordance with the terms of the Data Protection Act 1998.** ☐

Participant's Statement:

I _____agree that the research project named above has been explained to me to my satisfaction and I agree to take part in the study. I have read both the notes written above and the Information Sheet about the project, and understand what the research study involves.

Signed **Date**

Investigator's Statement:

I _____confirm that I have carefully explained the nature, demands and any foreseeable risks (where applicable) of the proposed research to the participant.

Signed **Date**

Appendix 12: Group comparisons at baseline when cases not meeting minimum response criteria are excluded

	Update	Exposure	Neutral	Statistical test
Age (median, IQR)	25.00 (3.00)	25.00 (5.00)	24.50 (3.00)	Kruskal-Wallis, $H(2)=0.026$, p=.99
Sex	8 males, 15 females	7 males, 23 females	6males, 16 females	Chi-squared, $X^2(2)=0.86$, p =.65

Baseline measures

	Update Mean (SD)	Exposure Mean (SD)	Neutral Mean (SD)	One Way ANOVA
STAI	35.00 (8.01)	36.48 (9.74)	32.82 (7.28)	$F(2,72)=1.01$, p=.37
PHQ-9	1.09 (1.68)	2.14 (2.52)	1.43 (2.11)	$F(2,72)=2.05$, p=.14
GAD-7	1.48 (1.75)	1.93 (2.15)	1.04 (1.70)	$F(2,72)=1.95$, p=.15
IES-R	6.70 (8.33)	8.03 (9.22)	6.13 (8.09)	$F(2,72)=.20$, p=.82
Trauma screener	2.43 (2.00)	1.85 (2.03)	2.28 (1.93)	$F(2,72)=.79$, p=.46

Data were log transformed prior to analysis. Untransformed values are reported.

Subjective distress ratings and SCR following acquisition

	Update	Exposure	Neutral	Statistical test
Distress rating CS+ACQ	55.87 (30.02)	43.35 (25.29)	40.00 (29.54)	One way ANOVA $F(2,72)=2.06$, p=.14
SCR CS+ACQ	0.199 (0.31)	0.2198 (0.44)	0.149 (0.42)	Kruskal-Wallis, $H(2)=0.54$, p=.76

SCR during acquisition films

	Update	Exposure	Neutral	Kruskal-Wallis
Film 1	0.41 (0.79)	0.41 (0.65)	0.36 (0.56)	$H(2)=1.30$, p=.52
Film 2	0.25 (0.48)	0.36 (53)	0.18 (0.47)	$H(2)=0.37$, p=.83
Film 3	0.53 (0.63)	0.45 (0.81)	0.42 (0.91)	$H(2)=0.32$, p=.85
Film 4	0.025 (0.21)	0.15 (0.39)	0.022 (0.07)	$H(2)=4.72$, p=.10
Film 5	0.33 (0.44)	0.38 (0.66)	0.23 (0.51)	$H(2)=0.91$, p=.63
Film 6	0.12 (0.47)	0.24 (0.68)	0.13 (0.55)	$H(2)=0.28$, p=.87

Self-reported diary compliance at follow-up

Diary	Update	Exposure	Neutral	One way ANOVA
Accurate	8.48 (1.20)	8.07 (1.25)	8.0 (1.71)	$F(2,72)=0.49$, p=.61
Reliable	2.04 (1.46)	2.24 (1.53)	2.09 (1.41)	$F(2,72)=0.11$, p=.89

Data were log transformed prior to analysis. Untransformed values are reported.

Service Evaluation Project

Depression in Children and Young People: How Compliant is SLAM with NICE Guidelines?

Supervisor: Dr. Patrick Smith

Abstract

Background: Depression in children and young people is consistently under-detected and has major costs both to the individual and society. The National Institute for Health and Clinical Excellence (NICE) provides guidelines for the treatment of depression in children and young people. Objectives: (1) identify who is presenting to South London and Maudsley NHS Foundation Trust (SLAM) with depression in terms of demographic factors and whether this is consistent with what would be expected from population and prevalence data. (2) Evaluate compliance of Child and Adolescent Mental Health Services (CAMHS) with NICE guidelines. Methodology: The Case Register Interactive Service (CRIS) was used to gather anonymised records from a one-year period which were (1) compared to prevalence and population data and (2) analysed and rated against NICE guidelines. Results: There is a large discrepancy between the expected number of cases and the number of cases seen by SLAM, especially in the 0 to 11 age group. Adherence to NICE guidance in terms of risk assessment and recording of comorbidities was good. Treatment of parental mental health conditions, use of a depression specific questionnaire, use of counselling/supportive therapy as a first line intervention and monitoring and use of medication are areas where adherence was poorer. Conclusions: The results indicate that a large proportion of children and young people suffering from depression are not being identified and treated. In general, adherence to NICE guidelines is good but there are areas that require improvement.

Contents

Figures

Tables

1. Introduction

1.1 Depression in children and young people

Depression in children and young people (C&YP) is characterised by a period of low mood and anhedonia with associated behavioural and cognitive symptoms. Behavioural features include fatigue and changes in sleep and appetite. Examples of cognitive symptoms are feelings of worthlessness, poor attention and low self-esteem.

Depression in C&YP has huge consequences for the individual and for society. It can lead to long-term social maladjustment with 37.5% experiencing social dysfunction in adulthood and this is exacerbated by comorbid conduct disorder (Fombonne et al., 2001b). Depression in C&YP has high recurrence rates (75.2%; Fombonne et al., 2001a) and is often undetected (Angold & Costello, 2001). It increases the risk of suicide (Fombonne et al., 2001b) and some suggest that it effects the chemical and physiological development of the brain resulting in long-lasting changes (Post, 1992; Sokolov & Kutcher, 2001).

Depression is associated with large economic costs. Depression in adults costs approximately £9,000 million each year in England (Thomas & Morris, 2003) and 30% of C&YP who experience depression in childhood will also experience depression as adults (NICE, 2005). However, there is very little research directly looking at costs of depression in C&YP. The economic burden of depression in childhood includes treatment costs, loss of productivity due to premature deaths and lost employment costs in terms of preventing parents from working and C&YP working in adulthood.

1.2 Prevalence and demographic factors

Studies estimate that, in a 12-month period, the prevalence of depression for pre-pubertal children is approximately 1% and rises to 3% after puberty (NICE, 2005). However, there is a lack of consistency in estimating prevalence rates and difficulties in reliably diagnosing depression (Carr, 2006).

Sex difference: research indicates that there is no sex difference in rates of depression in pre-pubertal children but that prevalence rises faster in females as they enter adolescence (NICE, 2005). After age 13, prevalence rates of depression in females are generally accepted to be

double that of males (Angold et al., 2002). Depression may present differently in males and females and some suggest that prevalence rates are equal for both sexes but that depression manifests itself differently in males (Cochran & Rabinowitz, 2000; Real, 1997). This argument highlights that men are at a consistently higher risk of suicide than women and suicide is the third leading cause of death among white males aged 15 to 24.

Sociocultural considerations: awareness and inclusion of social and cultural variables in the assessment and treatment of depression is a highly important but inherently complicated area. Whether depression presents and can be treated in the same way across different ethnicities and cultures is contentious (e.g. Ahmed, 2001).

There is some evidence that prevalence varies between ethnicities and with age and diagnostic categories. For example, boys aged 5 to 10 show similar rates of emotional disorder but Pakistani and Bangladeshi adolescents aged 11-15 show higher rates (approx. 12%) than white, black and Indian adolescents (approx. 5%)(Meltzer et al., 2000). The presentation and course of depression may also vary according to an interaction between culture and social situation. For example, Lau et al (2002) illustrated that a high risk factor for Asian American youths attempting suicide was a cultural conflict with their parents coupled with low levels of acculturation.

There is some suggestion that C&YP from ethnic minorities may have less access to mental health services but evidence is mixed. For example, some studies indicate a significantly lower rate of referrals by primary care for C&YP from Black and Minority Ethnic groups whilst others show referral rates to be higher (Malek & Joughin, 2004). Language, cultural conceptualisations and diversity in the manifestation of distress may present barriers for accurate assessment. Malek & Joughin (2004) highlight the importance of training and use of interpreters to aid assessment.

1.3 Assessment

Careful assessment of depression in C&YP is essential. Lack of detection of depression is a major problem with estimates that 75% of C&YP with a clinically identifiable mood disorder go undetected (Coyle et al., 2003). The clinical presentation of depression in C&YP varies considerably and the reasons for this are not well understood. Younger children tend to display more somatic symptoms, psychomotor agitation and separation anxiety whilst older children have more cognitive features such as worthlessness and self-criticism as well as

greater weight change and suicidal ideation (Ryan et al., 1987; Goodyer & Cooper, 1993; Kolvin & Sadowski, 2001; Luby et al., 2003).

Assessment instruments: NICE recommends routinely screening young people over 11 years old for depression using a self-report questionnaire. The Mood and Feelings Questionnaire (MFQ; Angold et al., 1995) is the most widely used and best validated screening instrument (NICE, 2005; Carr, 2008). NICE also recommends using the Strengths and Difficulties Questionnaire (SDQ) as a routine outcome measure. In terms of interviewer based tools, the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) and the Child and Adolescent Psychiatric Assessment (CAPA) have been shown to be reliable (Kaufman et al., 1997; Angold & Costello, 1995) and may improve accuracy in diagnosis. However, NICE suggests that the K-SADS and CAPA would need modification for use in CAMHS as they are time consuming.

Risk Assessment: Depression is associated with higher rates of suicide. When onset occurs in childhood, 32.3% of people with major depressive disorder (MDD) and 66% of people with MDD and comorbid conduct disorder will attempt suicide at some point in their lives (Fombonne et al., 2001b). There is a higher risk of suicide associated with C&YP with depression compared to other disorders. The assessment and management of suicide risk is therefore essential when considering C&YP with depression (Carr et al., 2008). Emotional disorders are also associated with an increased risk of deliberate self-harm (Green et al., 2004).

Comorbidity: Depression in C&YP rarely presents as a single disorder with 40-80% of cases meeting the criteria for another non-depressive disorder. Estimated rates of comorbid disorders include 25% of cases with comorbid conduct disorder and/or oppositional disorder, 25% with separation anxiety disorder and 15% with obsessive compulsive disorder (Goodyer & Cooper 1993; Herbert et al., 1996; Kovacs et al., 1989; Mitchell et al., 1988).

Environmental factors: Environmental factors are important to consider during assessment as 95% of C&YP experiencing a major depressive episode have long-standing psychosocial difficulties (NICE, 2005).

*Parental Mental health: 5*1% of parents of C&YP with an emotional disorder compared to 23% of parents of C&YP without an emotional disorder scored above cut off for an emotional disorder on the General Health Questionnaire (GHQ). Very high scores (9-12) on the GHQ were found in approximately one fifth of parents of children with an emotional disorder (Green et

al., 2004). NICE recommends that a family history should be taken to check for unipolar and bipolar depression in the family and that the parent's mental health should be treated in parallel.

1.4 Treatment

1.4.1 Psychological interventions

There have been a number of psychological therapies explored for treating depression in C&YP but the evidence base for most of these is extremely limited. The NICE review (2005) found that the psychological intervention with the largest evidence base was Cognitive Behavioural Therapy (CBT) then Interpersonal Psychotherapy (IPT) with a smaller evidence base for shorter-term Family Therapy (FT) and an even smaller one for individual psychotherapy. The review found that compared to control groups, psychological therapies produce greater gains during treatment and that these are maintained at follow-up. However, gains in the control group match the therapy group at one-year follow-up. Considering the importance of this time for a child or young person's social, cognitive and emotional development, the impact of accelerating the resolution of depression should not be underestimated. Therefore, NICE recommends a specific psychological therapy (CBT, IPT or short-term FT) as the first line treatment for moderate to severe depression.

1.4.2. Pharmacological interventions

The use of medication in C&YP with depression emerged from its evidence base in adults. However, a Cochrane review of tricyclic antidepressants (TCAs) indicated that they are no better than placebo for improving rates of recovery (Hazell et al., 2002). Selective serotonin reuptake inhibitors (SSRIs) have been linked to increased suicide risk in young people (e.g. Healy, 2003) and Fluoxetine is the only medication considered to have a positive balance of risks and benefits. A review by the Committee on the Safety of Medicines (CSM, 2003) suggested that the use of SSRIs was contraindicated for the treatment of depression in the under 18s. However, the CSM also specified that SSRIs other than Fluoxetine can be prescribed under some conditions, for example if drug treatment is indicated but Fluoxetine is not appropriate (NICE, 2005).

There is some evidence that combining fluoxetine and CBT may be most effective in reducing depressive symptoms and decreasing the risk of suicidal ideation associated with prescribing Fluoxetine alone (NICE, 2005). NICE does not recommend medication as a first line treatment

for depression and recommends that Fluoxetine should only be offered in combination with psychological therapy. In addition, due to the potentially increased suicide risk, regular monitoring is important.

1.5 National Institute for Clinical Excellence (NICE) guidelines

Studies on the NHS in the 1990s indicated great geographical variation in access to treatments and that new clinical evidence was only very slowly incorporated into routine care (Rawlins, 2004). Therefore, the Department of Health established NICE in 1999 to coordinate treatment guidelines on a national level. NICE produces guidelines for the treatment of individual medical conditions based on systematic reviews of clinical and cost effectiveness data. It is hoped that these guidelines will speed the transmission of evidence-based practice into clinics, improve standards of care for patients and reduce inequalities in access to treatments.

The success of monitoring and implementing NICE guidelines has been mixed. Monitoring compliance with NICE guidelines is complicated and it is unclear who is responsible for it:
"Responsibility for monitoring compliance is vague… (with) no clear lead role and often no objective measure of implementation." (Dent & Sadler, 2002)
This makes it difficult to estimate compliance and to target procedures to increase implementation. Adding to this, there are no clear rewards or sanctions for organisations that implement guidelines quickly or slowly. A recent audit of Assessment and Brief Treatment teams in South London and Maudsley NHS Foundation Trust (SLAM) (Rhodes et al., 2010) looked at the implementation of NICE guidelines for adults with depression. This audit found that guidelines were not being consistently implemented, particularly in terms of access to CBT. The successful implementation of NICE guidelines depends on organisational and individual support for them including available resources, knowledge and skills (Grimshaw et al., 2002). It is important to know whether the guidelines are being appropriately implemented due to the large costs involved in the development and introduction of the guidelines as well as monitoring the provision of best possible care.

1.6 Audit Aims

This audit consists of two main questions:

1.6.1. Part 1: Who is presenting to SLAM?

- Gather demographic and service information on the C&YP presenting to SLAM with depression in a one-year period.
- Compare the cases seen by SLAM to what would be expected according to population figures and prevalence rates.

1.6.2. Part 2: Is SLAM compliant with NICE guidelines?

- Evaluate the level of adherence in SLAM to NICE guidelines when treating C&YP with depression.
- Validate good adherence and highlight areas where adherence or recording of information is lacking.

2. Audit methodology

The Case Register Interactive Service (CRIS) allows authorised researchers regulated access to information from the electronic Patient Journey System (ePJS). CRIS was developed by SLAM and the Institute of Psychiatry (IoP) and enables the researcher to search through anonymised records from ePJS.

2.1 Defining the group

The group was defined by a number of terms including having an 'event' (an entry in the case notes on ePJS) between 14th June 2010 and 14th June 2011, having primary or secondary diagnosis that included the search string "depress*" and that they were seen in the CAMHS Clinical Academic Group (CAMHS CAG). Cases were excluded if the person was not being treated in CAMHS for depression e.g. parents of C&YP with depression. 862 cases with an age range of 6 to 21 were identified at this point ('original sample').

2.2 Part 1: Who is presenting to SLAM?

The expected number of cases of depression was estimated using population data from the Office of National Statistics (ONS) and prevalence data cited in NICE guidelines (2005). In order to compare the expected number of cases to the number of cases found by the search, the groups had to be defined by the same parameters. The ONS data was population estimate data from mid-2010 defined by borough and both ONS and NICE define the group as up to age 18. So, cases that were over age 18 or had a borough not known to SLAM were excluded to ensure that the sample data reflected ONS and prevalence data. 770 cases were identified for inclusion.

2.3 Part 2: Is SLAM compliant with NICE guidelines?

2.3.1 Defining the sample: matching the demographics of the group to the sample

The 'original sample' was stratified according to age, sex, ethnicity and borough and a random sample of 5% of cases (45 cases) was taken that matched the spread of data identified by the stratification.

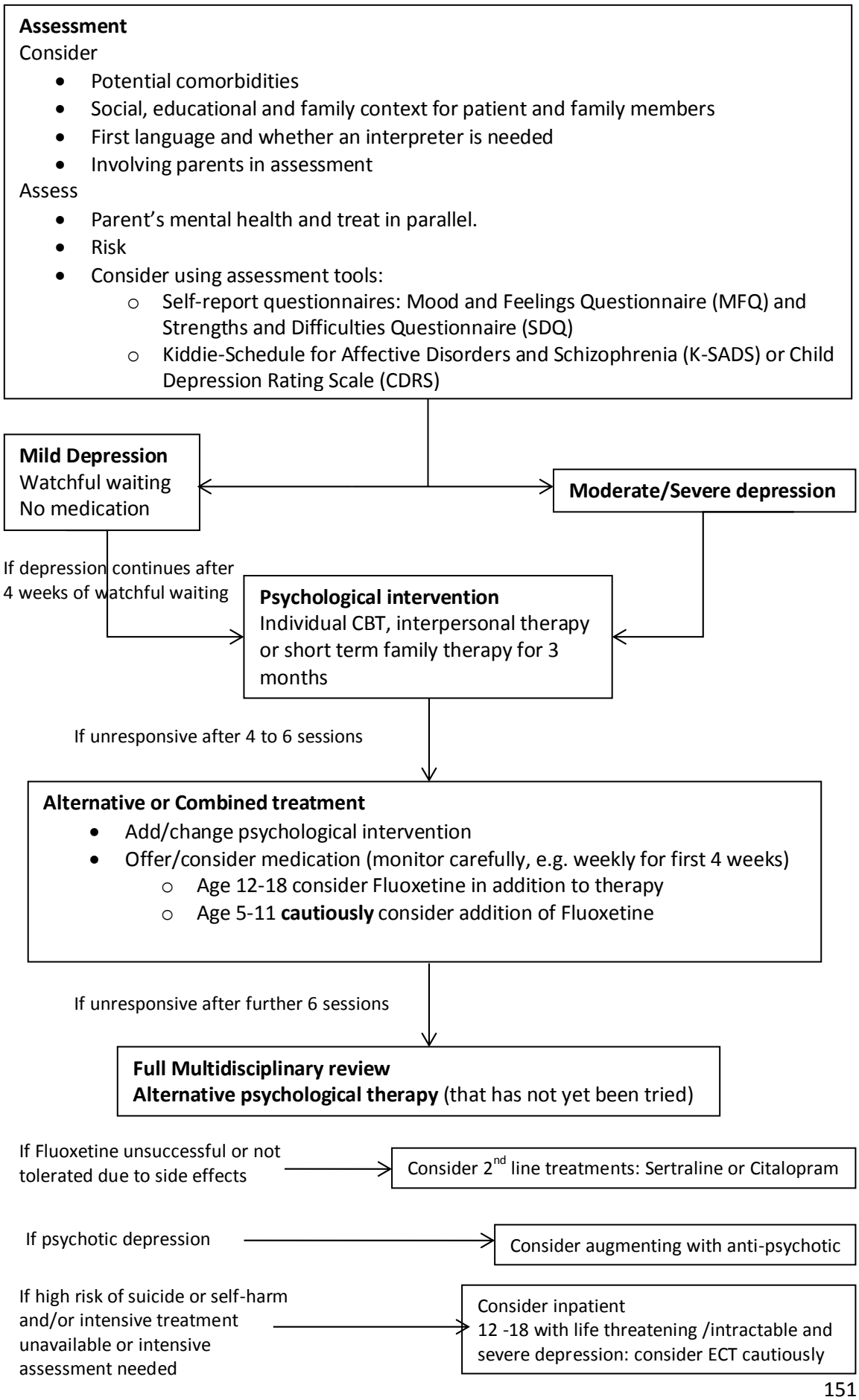
2.3.2 Adherence to NICE guidelines

NICE published clinical guidelines for the assessment and treatment of depression in C&YP in primary and secondary care in the UK in 2005. NICE makes recommendations across tiers 1 to 4 but this audit only considered recommendations made for tiers 2 to 4 as this was the data available. To evaluate adherence to NICE guidelines, specific guidelines were identified for consideration (Figure 1) and a search strategy was developed for each target guideline. This ranged from searching fields using the structure of ePJS to 'hand searching' through the event notes. For example, it is expected that a risk screen is entered in a specified field whereas there is no field for psychological therapy so this had to be identified by 'hand searching'. The search strategy for each guideline varied according to what could be reasonably expected to be entered into ePJS by the clinician.

2.4 Ethical approval

This project received Audit approval from the CAMHS Audit Committee and Information Governance Services and did not need ethical approval.

Figure 1: Summary of guidelines that are being audited.



3. Results

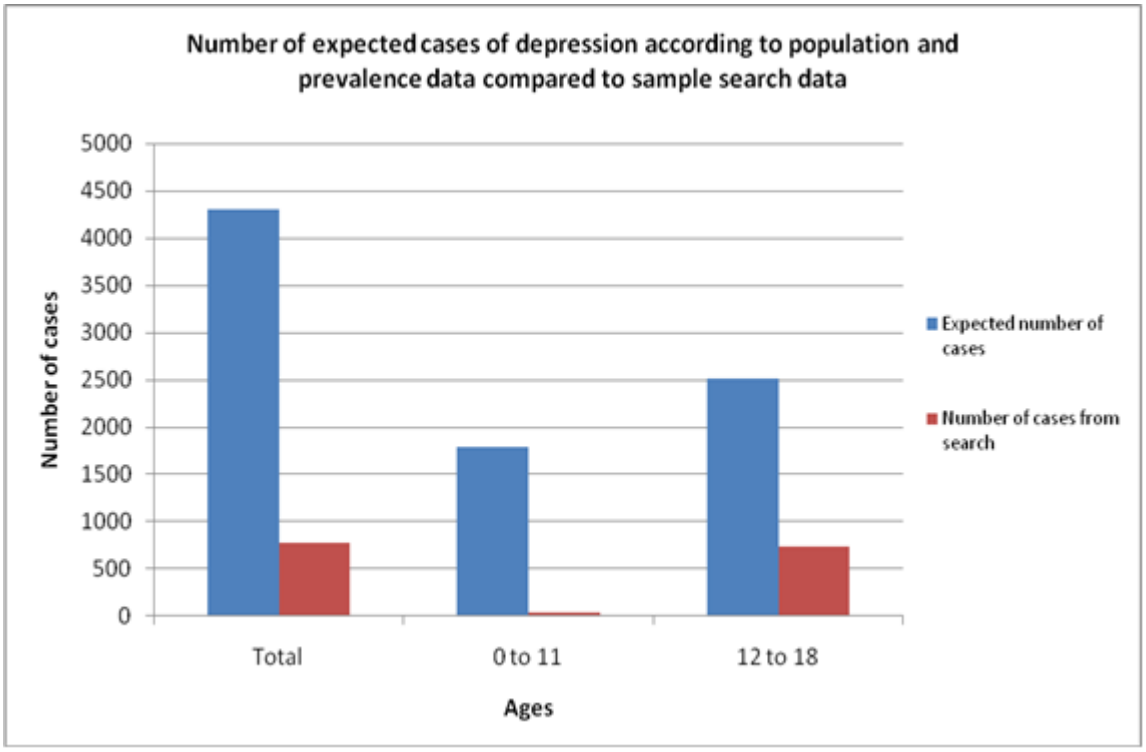
3.1 Part 1: Who is presenting to SLAM?

3.1.1 Number of cases

The 12-month period prevalence estimates for depression are approximately 1% for pre-pubertal children and 3% for post-pubertal children and adolescents (NICE, 2005). SLAM saw 2% of estimated cases of depression in the 0 to 11 age group and 29% of estimated cases in the 12 to 18 age group (Table 1 and Figure 2). In addition, the youngest case was 6 years old and so, no children aged 2 to 5 were seen in SLAM at the time of data collection.

Table 1 and Figure 2: Comparing expected number of cases of depression to sample data.

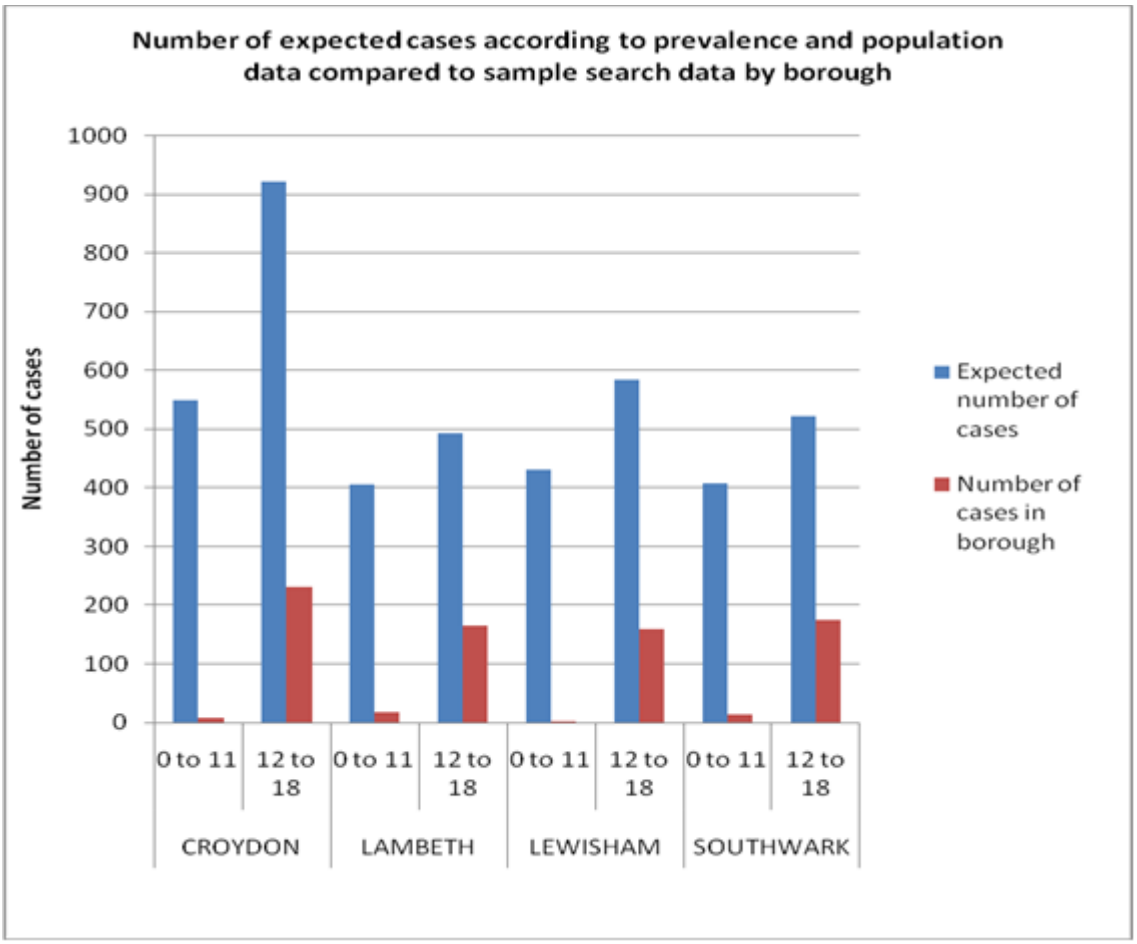
Age	Population	Expected number of cases	Number of cases seen by SLAM	Percentage of cases seen by SLAM
Total	263,100	4310	770	18%
0 to 11	179,136	1791	39	2%
12 to 18	83964	2519	731	29%



Number of cases by borough

Figure 3 illustrates the number of estimated cases of depression compared to the number of cases in the search within each borough. Each borough saw at least a quarter of the predicted number of cases in the 12 to 18 year group but all saw less than 5% of cases in the 0 to 11 age group. Croydon and Lewisham only saw 1.3% and 0.2%, respectively, of estimated cases in the age 0 to 11 group (Table A in Appendix 1).

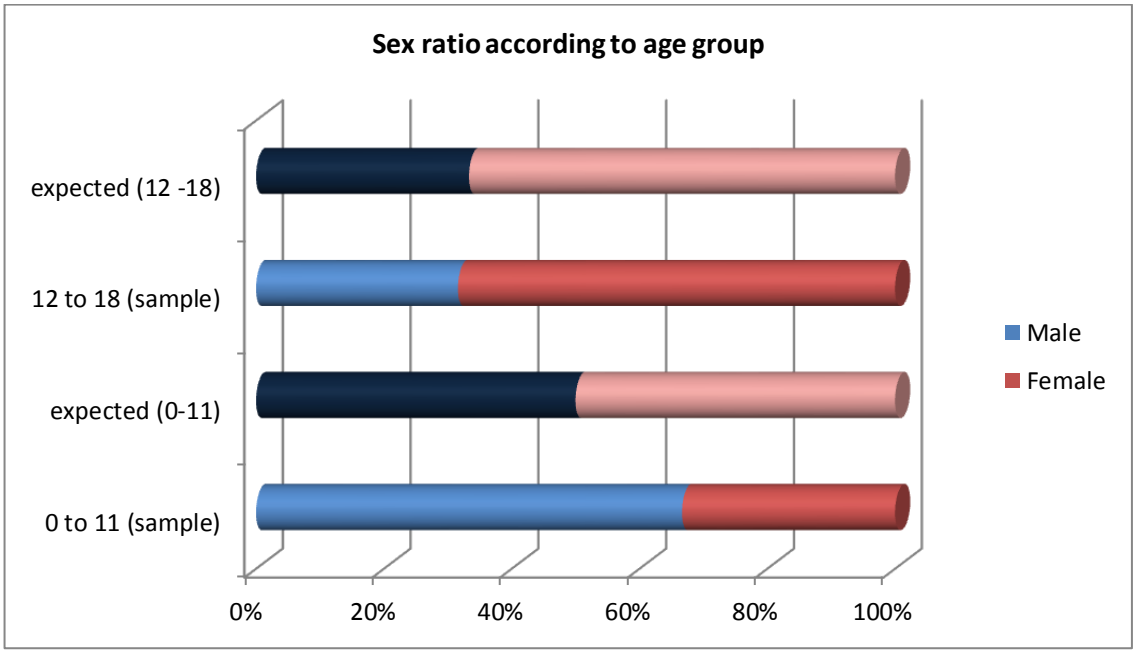
Figure 3: Number of cases by borough.



3.1.2 Sex ratio (all ratios quoted are female: male)

ONS data indicates a roughly equal number of male and female C&YP within the SLAM boroughs (Table B Appendix 1). The sex ratio in the sample would be expected to be 1:1 for the under 11s and approaching 2:1 for the 12 to 18 age group (NICE, 2005). The actual ratios seen were 1:2 in the under 11s and just over 2:1 in the 12 to 18 age group. (Table C in Appendix 1 and figure 4). This suggests that females in the 0-11 age group are being under-detected in SLAM.

Figure 4: Sex ratios.



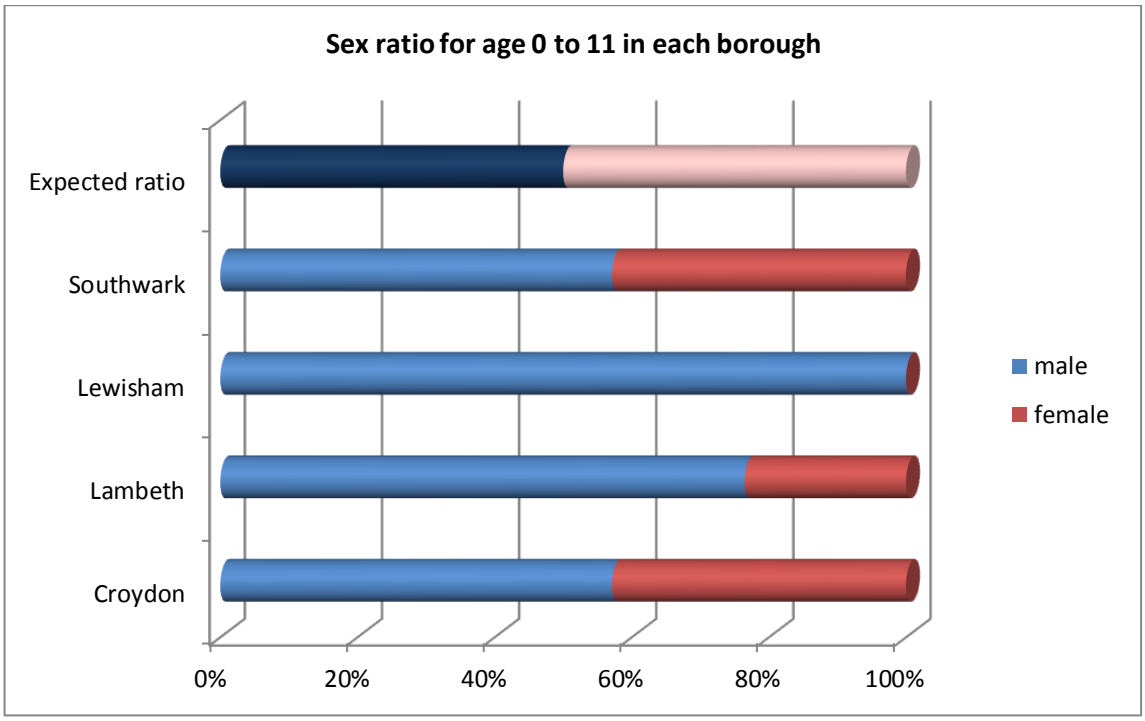
Sex ratios (female: male) in boroughs

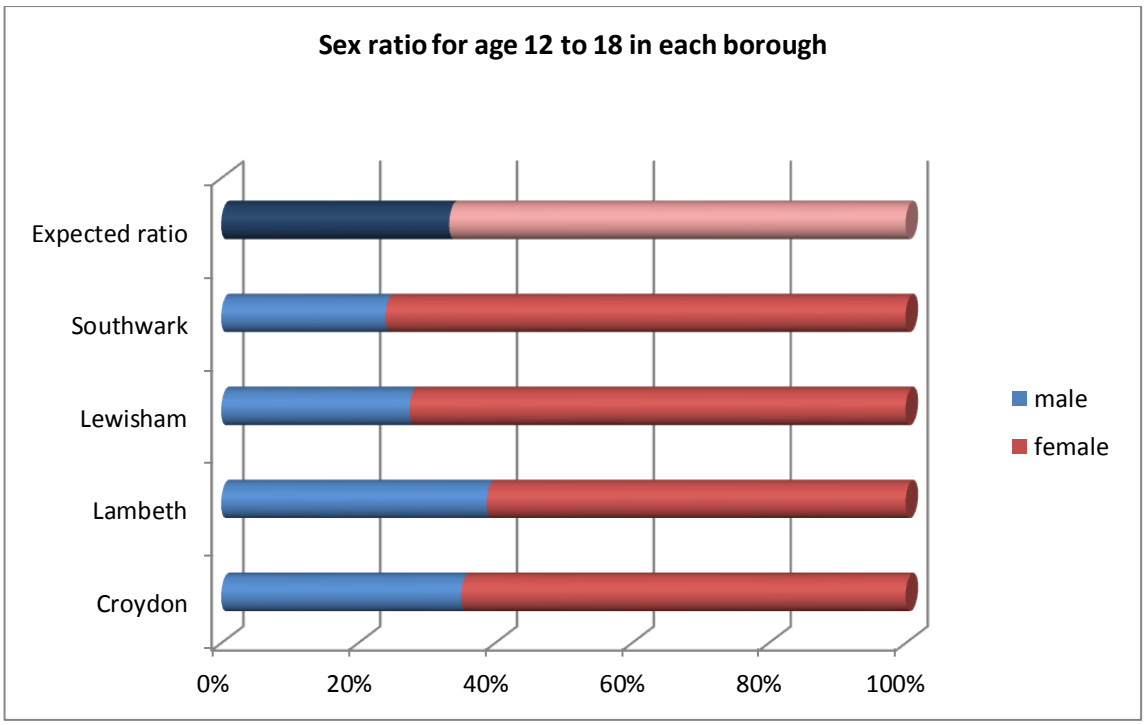
The ONS data also illustrates that there are equal numbers of males and females within each borough (see Table D in Appendix 1). In the 0 to 11 age group, Lambeth saw over 3 times the number of males to females so were under-detecting females in this age group (Figure 5 and Table E in Appendix 1). Lambeth was also the borough that saw the highest number of cases in the 0 to 11 age group. Lewisham only saw 1 case in the 0 to 11 age group in this one-year period and this case was male. The sex ratio for both Croydon and Southwark was 1:1.33. Therefore, the borough which is detecting more depression in the 0 to 11 age group (Lambeth), is primarily detecting depression in males.

In the 12 to 18 age group, the sex ratio varied depending on borough: 3.2:1 in Southwark, 2.6:1 in Lewisham, 1.9:1 in Croydon and 1.6:1 in Lambeth (Figure 6 and Table F in Appendix 1). So

this indicates that Southwark is significantly under-detecting males with depression aged 12 to 18.

Figure 5&6: Sex ratios in each borough for each age group.



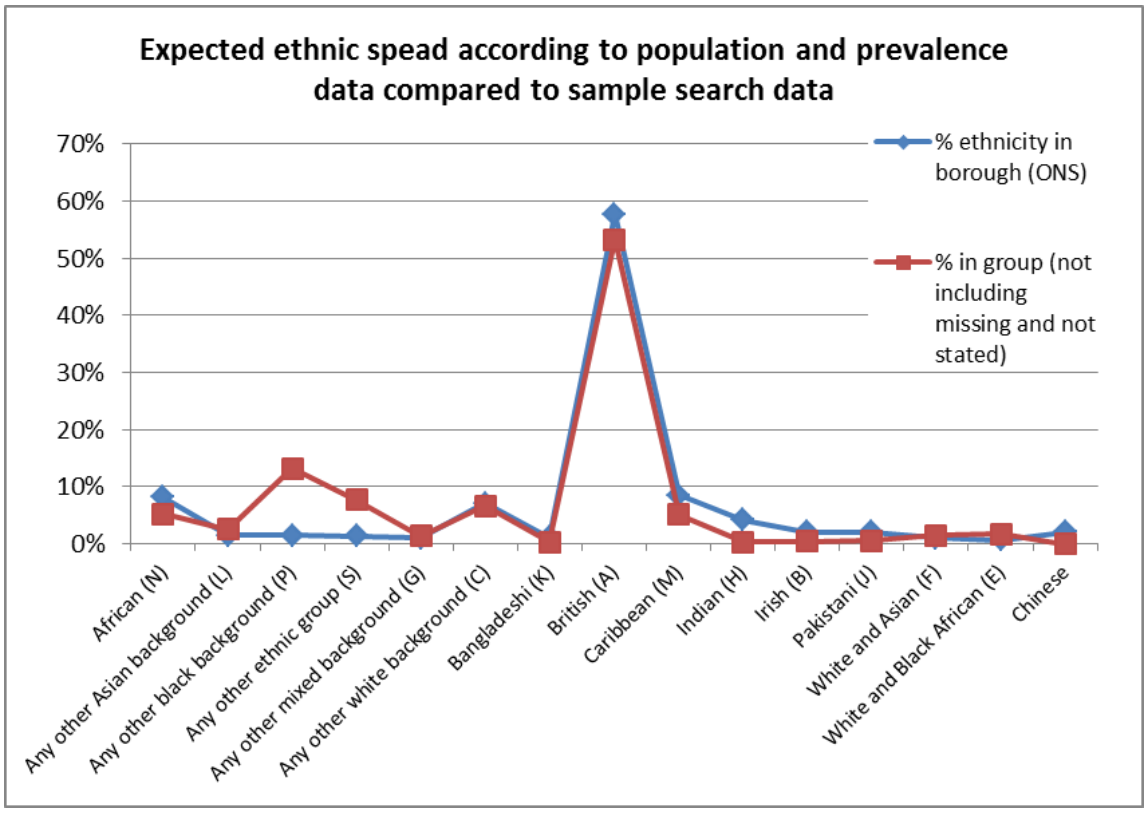


3.1.3 Ethnicity

The different ethnicities that make up the sample group were compared with the expected spread of the group according to adult population data as child ethnicity data was not available. This assumes that ethnicity within a borough is fairly independent of age and may have implications in terms of "any other groups" or mixed heritage groups (for example 'white and Asian' and 'white and black African'). There is some suggestion that prevalence rates of depression may vary according to ethnicity and that within ethnicities rates may vary depending on age and diagnostic category. However, evidence for this is mixed and the interaction between culture, presentation and incidence is unclear. Therefore, the data was not weighted according to whether an ethnic group may be more vulnerable to depression.

Figure 7 illustrates the percentages of different ethnicities across the boroughs and within the group. Some notable differences in the ethnic make-up of the population compared to the sample group (ONS vs. sample data) are that 'any other black background' (1.4% vs. 13.3%) and 'any other ethnic group' (1.4% vs. 7.7%) were over-represented whilst 'Indian' (4.2% vs. 0.3%) were under-represented in the sample (Table G in Appendix 1).

Figure 7: Ethnicity



Ethnicities within boroughs

Table 2 illustrates the expected percentages of different ethnicities using ONS data compared to the sample percentages for each ethnicity within each borough with discrepancies of more than 5% highlighted in yellow (for graphs, see figures A-D in Appendix 1). These results reflect those found in the overall sample with 'any other black background' and 'any other ethnic group' being consistently overrepresented in the sample data. In addition, 'British' was under-represented in the Lambeth and Southwark samples and 'Caribbean' was under-represented in the Croydon and Lewisham samples. There may also be an under-representation of some of the minority ethnic groups such as 'Indian', 'Pakistani' and 'Chinese' (highlighted in grey). However, these groups represent a small portion of the sample which makes meaningful interpretation difficult due to large confidence intervals.

Table 2: Ethnicity data by borough.

	Croydon		Lambeth		Lewisham		Southwark	
	ONS	**Sample**	**ONS**	**Sample**	**ONS**	**Sample**	**ONS**	**Sample**
African (N)	5.95%	1.50%	8.40%	5.00%	8.69%	3.42%	10.15%	11.56%
Any other Asian background (L)	1.87%	2.50%	1.20%	1.88%	1.47%	3.42%	1.26%	2.31%
Any other black background (P)	1.08%	10.00%	1.52%	15.00%	1.70%	15.07%	1.54%	13.87%
Any other ethnic group (S)	1.08%	6.50%	1.38%	10.63%	1.40%	5.48%	1.79%	8.09%
Any other mixed background (G)	1.05%	2.00%	1.06%	0.63%	1.06%	2.05%	1.05%	0.58%
Any other white background (C)	5.49%	3.50%	8.65%	11.25%	6.80%	7.53%	7.84%	5.20%
Bangladeshi (K)	0.82%	0.00%	1.13%	0.63%	0.94%	0.00%	1.54%	0.58%
British (A)	60.42%	64.50%	56.78%	42.50%	56.95%	56.16%	56.06%	47.98%
Caribbean (M)	8.09%	2.50%	9.00%	8.13%	10.24%	4.11%	6.86%	6.36%
Indian (H)	5.90%	1.00%	3.60%	0.00%	2.87%	0.00%	4.03%	0.00%
Irish (B)	1.69%	0.50%	2.08%	0.00%	2.42%	0.68%	2.03%	0.58%
Pakistani (J)	3.21%	2.00%	1.66%	0.00%	1.44%	0.00%	1.58%	0.00%
White and Asian (F)	1.20%	2.50%	0.92%	1.88%	0.87%	0.68%	0.88%	0.58%
White and Black African (E)	0.55%	1.00%	0.71%	2.50%	0.72%	1.37%	0.70%	2.31%
Chinese	1.61%	0%	1.91%	0%	2.42%	0%	2.70%	0%

3.2 Part 2: Is SLAM compliant with NICE guidelines?

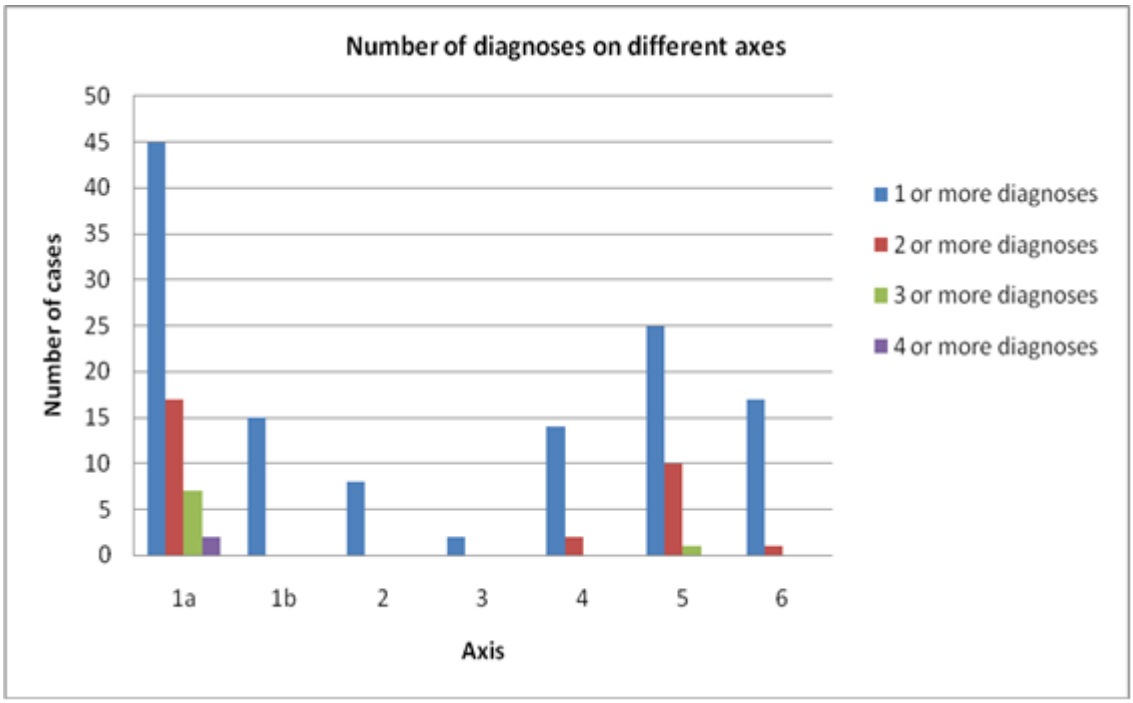
45 cases were identified (5% of original sample) by random sampling. This group reflected the demographic spread of the original sample.

3.2.1 Assessment

Comorbidities

42 of the 45 cases had a primary diagnosis (Axis 1a) and three had a secondary diagnosis (Axis 1b) that included the 'depress*' term. Over 70% of the cases had at least one additional diagnosis. 55% of the sample had at least one diagnosis on Axis 5 (associated abnormal psychosocial situations), 31% had a diagnosis on Axis 4 (additional medical conditions) and 18% had an additional diagnosis on Axis 2 (specific disorder of psychological development) (Figure 8).

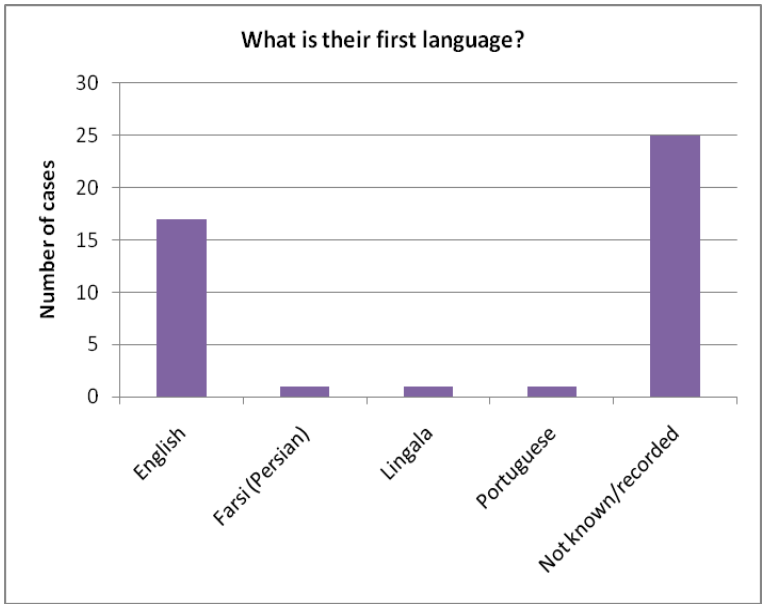
Figure 8: Comorbidity

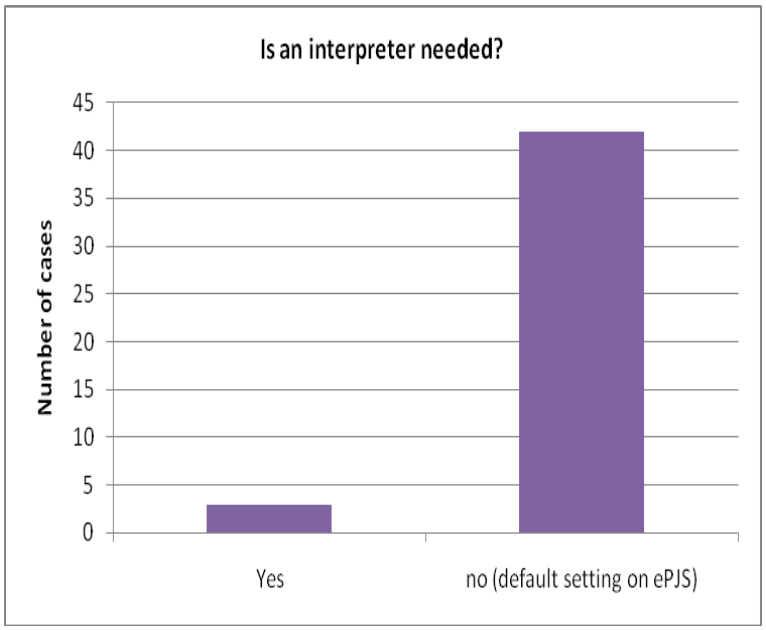


Language

In 55% of the cases the client's first language was not recorded (Figure 9) with the majority of those that were recorded being English. Over 90% of cases were recorded as not requiring an interpreter (Figure 10). However, this is the default setting on ePJS.

Figures 9 and 10: First language and requirement for an Interpreter

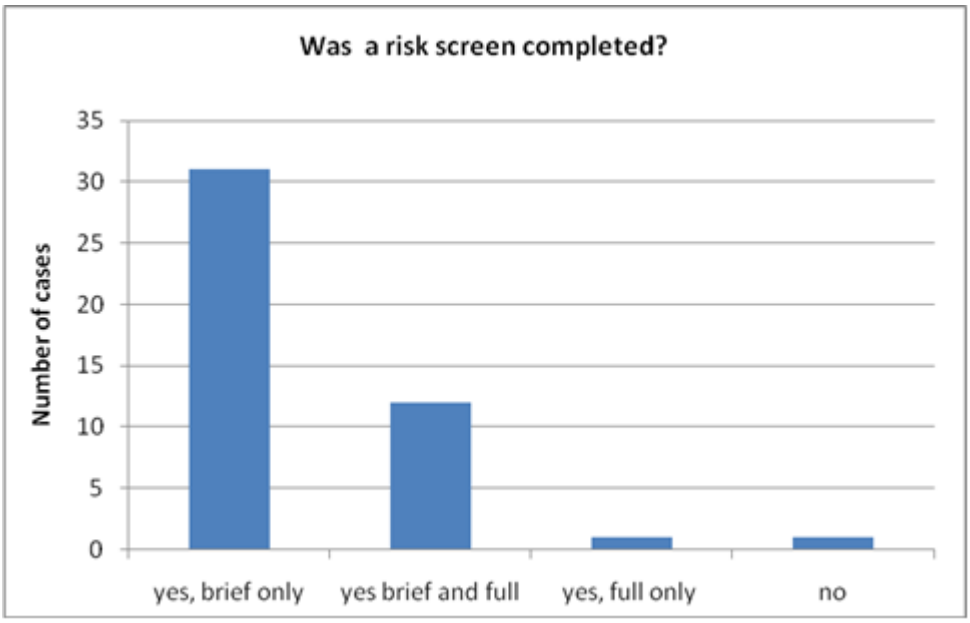




Risk Assessment (Guideline 9.3.3)

96% of the cases had a risk screen appropriately completed. One case did not have a completed risk screen and one had a full screen completed without a brief risk screen. 29% of the sample had a full risk screen completed (Figure 11).

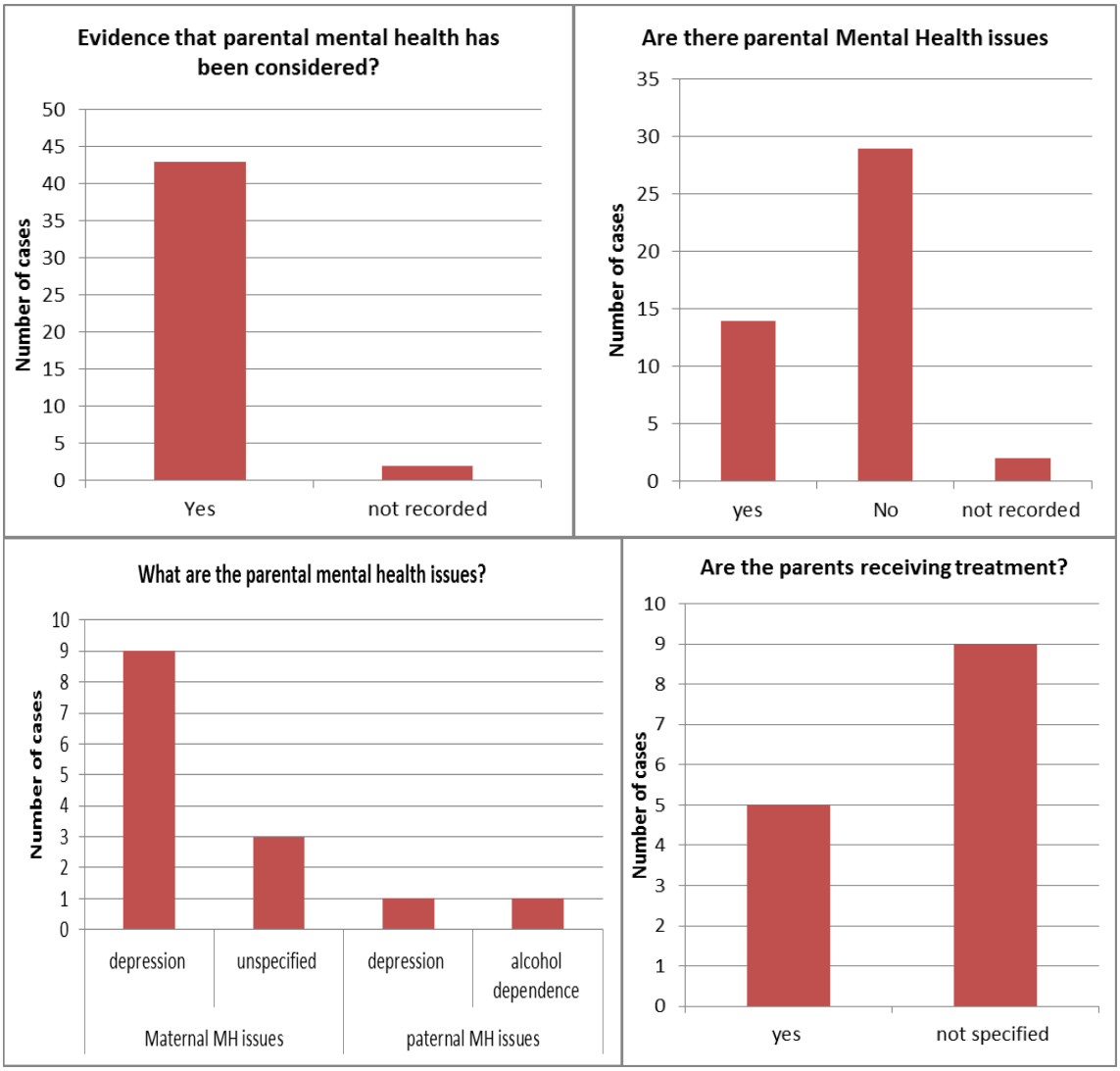
Figure 11: Risk screen



Parental Mental Health

In 96% of the cases, there was evidence that parental mental health for at least one parent had been considered (Figure 12). Of these, 33% had mental health issues (Figure 13) with the majority (86%) being maternal mental health issues, mostly with a history of depression (75% depression, 25% unspecified). There was a paternal mental health problem recorded in two cases: 1 was depression and 1 was alcohol dependence (Figure 14). In 65% of the cases, it was not specified whether the parents were receiving any treatment for their mental health problems (Figure 15).

Figures 12 to 15: Parental mental health

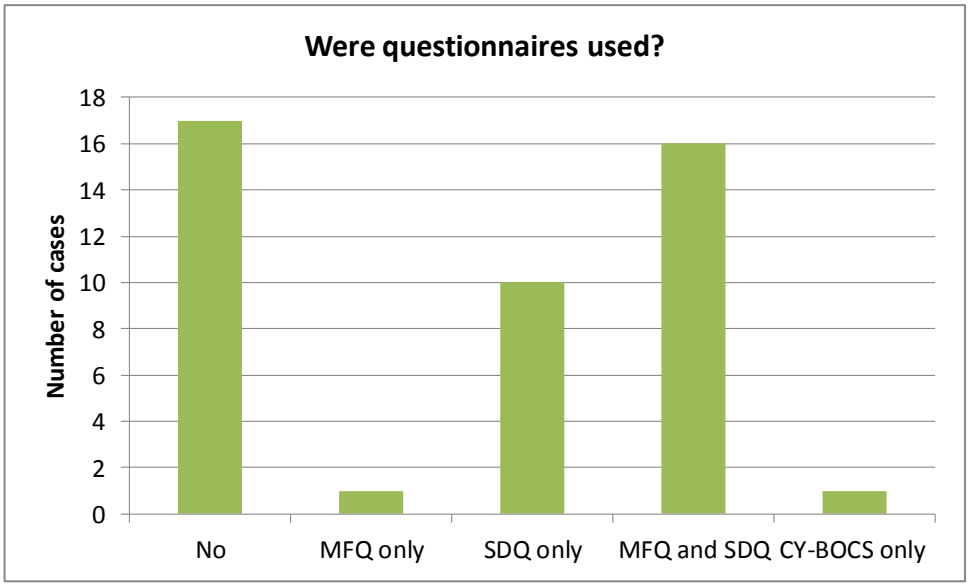


Assessment tools

There was evidence that a questionnaire was used in 62% of cases (Figure 16). In 36% of cases, both the MFQ and the SDQ were used. 58% of cases used the SDQ and 38% of cases used the

MFQ. Additional questionnaires used included those assessing anxiety disorders, for example Obsessive Compulsive Disorder and Post-Traumatic Stress Disorder (e.g. CY-BOCS, CPSS and the SCARED). NICE guidelines recommend that routine screening includes the MFQ for children over the age of 11. Only one child in the sample was under 11 and therefore, 61% of cases did not receive the appropriate self-report questionnaire.

Figure 16: Use of questionnaires



3.2.2 Treatment

Psychological intervention

69% of cases were offered a psychological intervention (Figure 17) by the team treating them. The remaining cases were either not offered intervention, were referred elsewhere or it was unclear from the notes. 24% were clearly not offered a psychological intervention and the most frequent reason (55%) was because they were offered counselling/supportive therapy instead (Figure 18).

Figure 17: Cases offered psychology

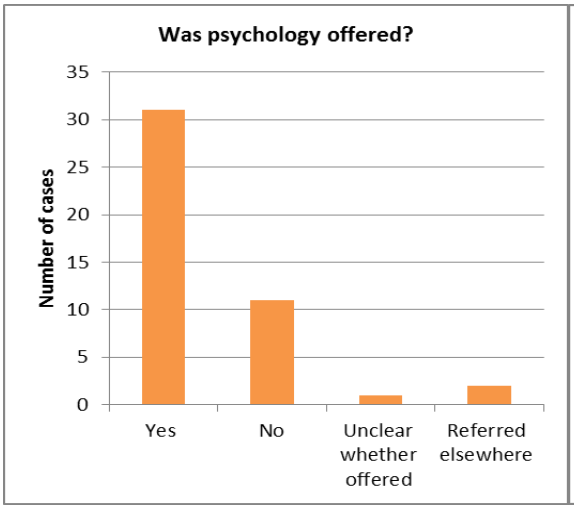
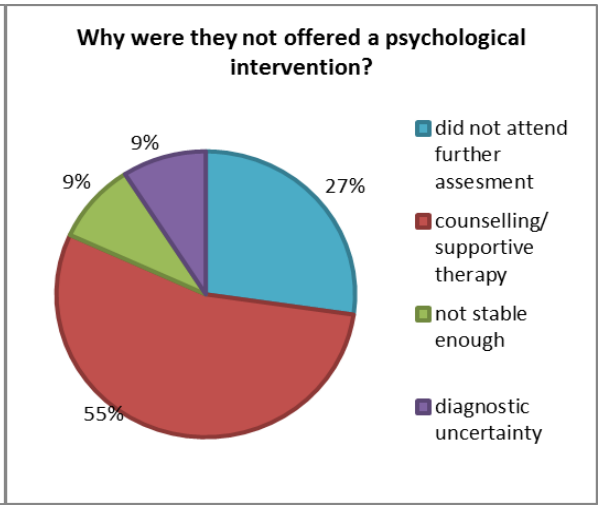
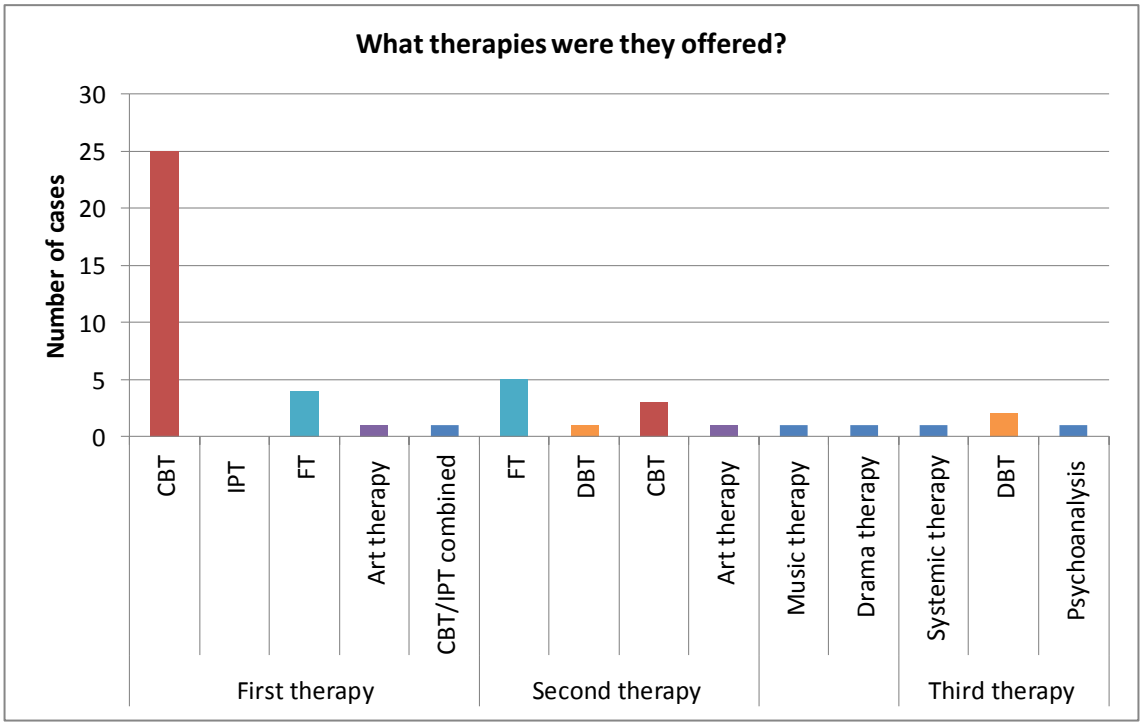


Figure 18: Reasons for not offering psychology



Of those that were offered psychological intervention, 84% accepted (Figure E in Appendix 1). Of those that received an intervention, 61% received one psychological therapy, 26% received two and 13% received three (Figure F in Appendix 1). The most common first line psychological treatment was CBT (81% of cases) and Family Therapy was the most common second line psychological treatment (40% of cases) (Figure 19). No cases received IPT alone.

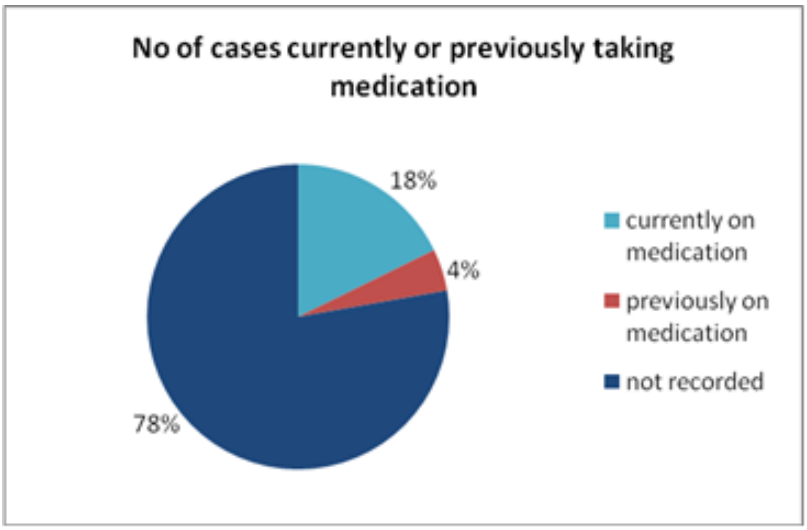
Figure 19: Psychological therapies offered.



Medication

22% of the cases were recorded as having previously taken or currently taking medication (Figure 20).

Figure 20: Medication



Of those 10 cases that were prescribed medication, 60% were prescribed Fluoxetine (capsules or oral suspension) as their first medication (Figure 21). 50% of the cases were currently taking one medication and 30% currently taking two different medications (Figure G in Appendix 1). 40% of the cases were offered medication before there was evidence that they were offered psychological therapy (Figure 22). The case that was prescribed Risperidone as the first line treatment had an additional axis 1 diagnosis of 'unspecified non organic psychosis'. The case that was prescribed Aripiprazole had a diagnosis of 'severe depressive episode without psychotic symptoms' and the case that was prescribed Amitriptytyline had diagnoses of 'moderate depressive episode' and 'childhood autism'.

Figure 21: First medication offered.

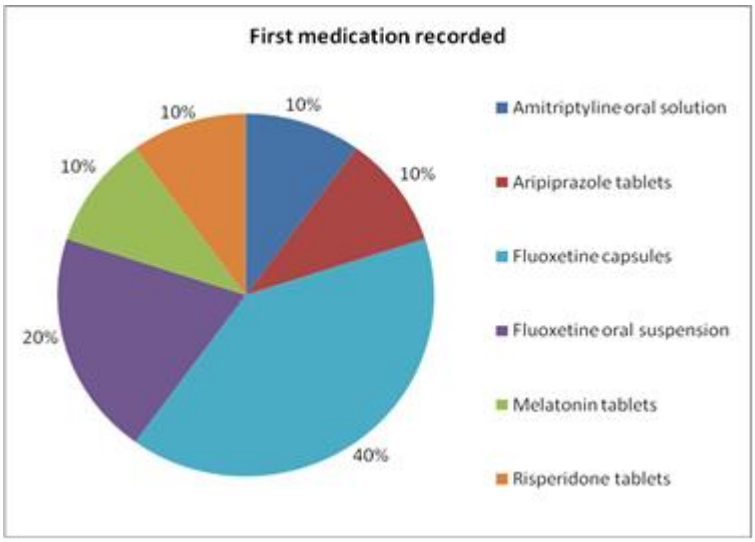
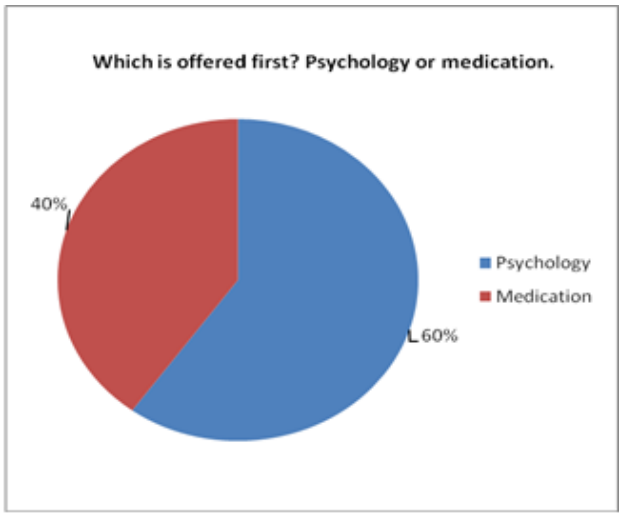
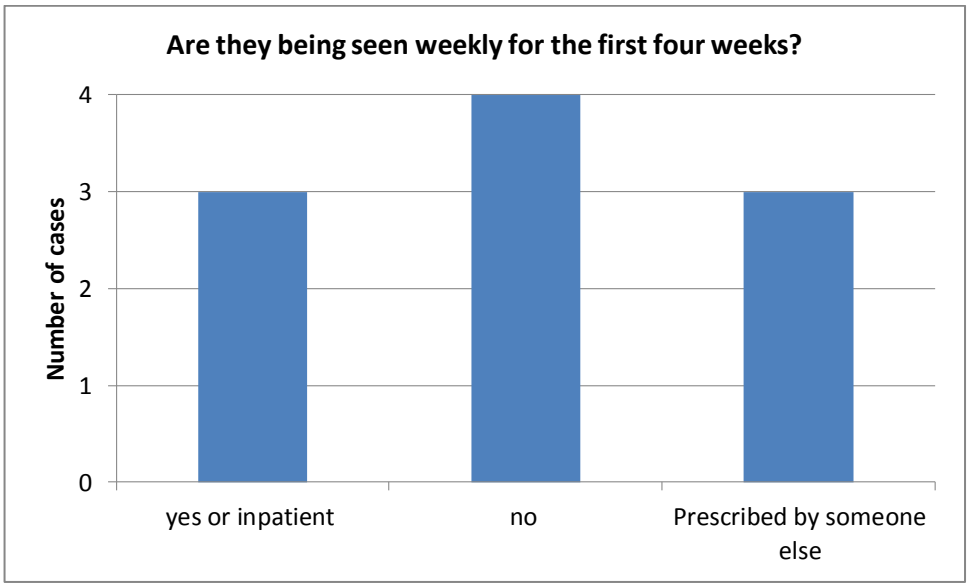


Figure 22: First line treatment.



In four cases, there was no evidence that they were seen weekly by CAMHS when they were first prescribed medication. Only one outpatient case was seen weekly by SLAM (Figure 23).

Figure 23: Contact after prescription of medication.



4. Conclusions

This audit firstly compared the group of C&YP that SLAM were treating for depression with the group that would be expected to present to SLAM with depression in a one-year period. Secondly, this audit aimed to evaluate how compliant SLAM is with NICE guidelines for treating depression in C&YP.

4.1 Part 1: Who is presenting to SLAM?

There is a large discrepancy between the expected number of cases and the number of cases seen by SLAM. These findings suggest that a large proportion of C&YP with depression in Croydon, Lewisham, Lambeth and Southwark are not being identified and treated. Most pertinently, there appeared to be a very low number of 0 to 11 year olds being treated in CAMHS with figures indicating that CAMHS saw only 2% of estimated cases of depression in this age group.

In the under 11 age group, there were twice as many boys compared to girls seen across SLAM and three times as many seen in Lambeth. In the 12 to 18 group, Southwark saw significantly more females compared to males than would be expected. In terms of ethnicities, some groups appeared to be over-represented ('any other black background' and 'any other ethnic group') and others under-represented (e.g. 'Indian') in the sample.

4.2 Part 2: Is SLAM compliant with NICE guidelines?

Areas where compliance was good included the recording of additional diagnoses and risk assessment. Areas where compliance was lacking included the recording of first language, parental mental health assessment and treatment, use of a depression specific questionnaire, use of counselling/supportive therapy as a first line intervention and monitoring of medication.

In terms of assessment, the rates of comorbidity with another non-depressive disorder were high (70%) and in keeping with what would be expected from epidemiological studies. Over half of the sample had diagnoses on Axis 5 which highlights the importance of environmental stressors. NICE guidelines around risk assessment appear to be adhered to, with 96% of cases

having an appropriately completed risk screen. 29% required a full risk screen which gives some insight into the level of risk CAMHS are managing.

Parental mental health is vital to consider in the assessment and treatment of C&YP with depression and, in most cases, it had been recorded for at least one parent. A parental mental health issue was recorded in a third of cases which is lower than expected (Meltzer et al., 2000; Green et al., 2004). The vast majority of mental health problems recorded were on the maternal side and it is unclear whether this is due to lack of prevalence or lack of assessment of paternal mental health. Further to this, in 65% of cases where parental mental health difficulties were identified, it was not recorded whether the parent was receiving treatment. This is not in keeping with NICE guidelines.

A substantial proportion of cases (38%) did not complete a screening questionnaire. An even larger proportion (61%) did not complete a depression-specific questionnaire as recommended by NICE. Self-report and parent-report questionnaires are useful to aid assessment, diagnosis and to measure progress and outcome and therefore their routine use needs to be increased. First language was often not recorded on ePJS (55% of cases) and this should be increased. In 90% of cases it was recorded that an interpreter was not required. However, as this is the default setting on ePJS, it seems likely that this may not have been an active choice by the clinician. Therefore, the default setting should be changed to “not known” to avoid assumptions and allow meaningful data collection.

Treatment that was considered by this audit was medication and psychological intervention. Over two-thirds of cases (69%) were offered a psychological intervention with the first line treatment most commonly being CBT (81%) and family therapy the most common second line intervention (40%). No cases received IPT alone which is an unexpected finding as IPT is recommended by NICE as a first line treatment for moderate to severe depression. 24% of cases were not offered a psychological intervention. The most common reason for this (55% of cases) was that they were offered counselling or supportive therapy instead. This is not recommended by NICE guidelines as an effective treatment and needs to be addressed by services.

Only a small sub-group received medication (22%) which, given the guidelines that this should be a second line treatment, is encouraging. However, there were cases that were not

prescribed Fluoxetine as the first line treatment despite having a depressive disorder as their primary diagnosis. There was also evidence that medication was prescribed without psychological intervention and without monitoring over the first four weeks. This is clearly concerning especially considering evidence that SSRIs increase the risk of suicide in C&YP and that this risk can be reduced if administered in combination with CBT. However, it is difficult to comment further due to the small sample size.

5. Discussion

5.1 Part 1: Who is presenting to SLAM?

Depression is a source of serious distress and impairment as well as a leading risk factor for suicide in C&YP (Brent et al., 1993). This audit clearly indicates that the number of C&YP being treated for depression in CAMHS is substantially lower than would be expected from prevalence rates, particularly in the age 0 to 11 group. This may be due to a number of reasons including lack of presentation to services, lack of recognition of depression by services and lack of transfer of cases from tier 1 to tier 2. This raises significant questions around service provision and commissioning of services. The results from this audit are in line with studies that indicate under-treatment of C&YP with depression (Wu et al., 2001; Saxe, Cross & Silverman, 1988).

Factors associated with under-treatment include a failure to recognise symptoms, stigma and difficulties accessing services. Wu et al (2001) illustrated that parental recognition of symptoms and knowledge about depression was associated with the child receiving services. A child's access to services is more complicated than an adult's as they rarely seek help for themselves. Therefore, the systems around the child (school, family, after-school organisations) are crucial in aiding the identification of depression. Green et al (2004) found that 73% of parents of C&YP who were assessed as having an emotional disorder had sought help, most commonly from teachers (43%). However, only 24% of these parents contacted or were referred to mental health services. This indicates that there is a gap in parents accurately raising concerns about their child and being seen by mental health services. In addition, common obstacles identified by concerned parents who did not seek help were: believing a specialist would not be able to help (8%), being unaware of available services (7%) or struggling to get referred (5%).

There were no cases in the pre-school age group (2 to 5) in our sample. Studies have shown that depression is present in preschool children (e.g. Keenan et al., 1997; Lavigne et al., 1996) and that only a small proportion who meet criteria are referred to treatment (e.g. Lavigne et al., 1998). Diagnosing depression in pre-school children raises theoretical and ethical issues including the rapid and heterogeneous development of children in this age group, the stigma involved in giving a child a diagnosis and that by diagnosing, you may be locating the problem

within the child rather than their environment. Open discussion about these issues, increased awareness of depression in pre-school children and treatment pathways might lead to enhanced understanding and identification. Modifications could also be made to diagnostic criteria to aid identification (e.g. Luby et al., 2002).

In the under 11 age group, there was double the number of males to females in the sample. This is not expected as prevalence studies indicate a roughly equal sex ratio in pre-pubertal children. One possible explanation might be a difference in presentation between the sexes with boys being more likely to show externalising symptoms than girls. Parents and professionals may be more likely to identify a problem and seek help for boys if they are being disruptive at home or at school. Evidence suggests that C&YP are less likely to receive treatment for depressive compared to disruptive disorders (Cohen et al., 1991; Wu et al., 1999).

Whether ethnicity has an impact on depression being correctly identified and treated in SLAM is a complicated question and beyond the scope of this audit. It appeared that there were some ethnic groups that were over or underrepresented in the sample. This could be due to several factors including cultural narratives around mental health or misreporting of ethnicities on ePJS. The second reason seems probable given the overrepresentation of 'any other black background' and 'any other ethnic group'. However, it may also be that, because adult data was used to calculate expected ethnicities, more C&YP are self-defining in these groups compared to adults.

5.2 Part 2: Is SLAM compliant with NICE guidelines?

In general, adherence to NICE guidelines was good. However, there are some areas where it was lacking and this needs to be addressed. There was limited evidence that parental mental health issues were appropriately assessed or treated. Depression starts earlier and is associated with poorer prognosis in C&YP who have parents with an affective disorder (Emslie et al., 2003) and maternal depressive symptoms impact on treatment efficacy (Brent et al., 1998). In most of the cases reviewed, only one parent's mental health status was recorded (usually maternal). Both parents' mental health will impact on the child and should inform formulation even if only one is directly involved in care-giving. Therefore, the lack of full assessment and lack of parallel treatment of parental mental health difficulties is concerning as it has proven impact on the child's prognosis and successful treatment.

This raises service level questions about how to assess parental mental health and who should provide treatment for the parents. The General Health Questionnaire (GHQ, Goldberg, 1972), Patient Health Questionnaire (PHQ-9, Kroenke, Spitzer & Williams, 2001) or the Generalised Anxiety Disorder Assessment (GAD7, Spitzer et al., 2006) could provide quick and quantitative measures of parental mental health. In addition, a clear referral pathway and closer communication between CAMHS and IAPT should be established so that parental and child mental health can be treated in parallel.

In terms of comorbidity, the NICE review highlighted that 95% of cases of depression have long standing psychosocial difficulties. This audit found that 55% of the cases have a diagnosis on Axis 5 (associated abnormal psychosocial situations). This may highlight a recording issue or represent a difference in the definitions of 'psychosocial situations'. It seems likely that criteria on Axis 5 are more stringent than the definitions used by the NICE review. Psychosocial stressors clearly have a large impact on the development, maintenance and trajectory of depression and this highlights an area of inconsistency that needs to be addressed for clinical and research reasons.

One unexpected finding was that no cases received IPT alone despite it being a recommended first line treatment. This could be for a number of reasons including lack of knowledge and awareness about IPT and its evidence base, lack of trained IPT therapists or that C&YP with depression prefer other therapies. It would be interesting to investigate the reasons for this further and important to address them in order to ensure the most appropriate and valid psychological interventions are being offered.

This audit does raise two service and guideline –related questions. The first is around what can be usefully and realistically recorded on ePJS in terms of the limited time available to clinicians and how this time is best spent. Meticulous ePJS recording is time consuming and a compromise needs to be found between recording information and clinical time. There may be ways of changing the format of ePJS to make it more clinician-friendly and less time consuming. The second issue surrounds the complexity of cases being seen in CAMHS in terms of comorbid psychiatric and medical diagnoses and social adversity. There is a lack of research and clinical guidance for managing and treating these complex and often high risk cases.

Therefore, competent clinical judgment is essential and this should be reflected in the guidelines.

5.3 Limitations of the audit

In this audit, we only had access to clinical records from secondary care and therefore do not know how many C&YP are being assessed and diagnosed with depression in primary care. NICE guidelines recommend four weeks of watchful waiting for a C&YP presenting with mild depression. However, at the time of data collection, there was no provision for treatment of depression in C&YP in primary care. So, if the depression lasted for longer than four weeks or was more severe, these cases should have been referred to CAMHS.

A second limitation is that, Whilst CRIS provides a useful tool to access patient records confidentially and without reliance on clinician report, it does rely on correct recording and use of ePJS. Before beginning the audit, we decided that we could expect certain fields on ePJS to be completed and that if they were not, we would not hand search the records. This decision was made due to time constraints. Examples of this included the diagnostic and medication fields and so if they were not filled in, then it was assumed that the case did not have a diagnosis of depression or was not receiving medication. However in other areas where there are no dedicated fields on ePJS, hand searching was necessary, for example psychological therapy and contact for first four weeks of medication. This may have led to adherence being over or under estimated in some cases.

5.4 Limitations of NICE guidance

The power and purpose of NICE guidelines is to quickly translate research findings into clinical practice. However, research is usually carried out on a limited range of C&YP in terms of age, comorbidity, socio-economic status and severity of symptoms. Cases that are seen by CAMHS are often complex and rarely have a single diagnosis. Therefore, careful case formulation and clinical judgement is essential. There are a limited number of guidelines available covering other disorders or comorbidities in C&YP and this presents an area of limitation.

6. Recommendations

6.1 General

1. Dissemination of audit findings through written reports, presentations at meetings and discussion with professionals.
2. Consideration of ways to make guidelines more accessible and relevant to CAMHS professionals, for example having a brief summary of the key guidelines and having meetings with CAMHS staff to discuss the guidelines and why they are important.

6.2 Part 1: Who is presenting to SLAM?

1. Increased recognition and assessment of depression across age groups, especially in the under 11 age group. Consideration is required concerning which combination of teams would deliver the following:
    a. Psycho-education around the symptoms and treatment of depression in schools and tier 1.
    b. Increasing parental awareness of emotional difficulties in C&YP to enhance early identification and treatment, for example leaflets targeted at parents and placed in GP surgeries and schools.
2. Increased understanding by professionals of the different presentation of depression in males and females, for example through time allocated to Continued Professional Development.
3. Further investigation of referral patterns for Black and Minority Ethnic groups, for example through future audit.

6.3 Part 2: Is SLAM compliant with NICE guidelines?

1. ePJS
    a. Clear guidance on mandatory fields in ePJS and rationale for these fields e.g. impact of parental mental health on outcomes.
    b. Consider functionality within ePJS to remind clinicians when key fields are not completed, for example automated reminders or highlighted boxes.

   c. NICE guidance integrated more fully into ePJS e.g. flow chart for NICE recommendations when depression diagnosis is entered.

2. Parental mental health
   a. More integrated treatment of adult and child mental health issues. For example, direct referral pathways and heightened communication between CAMHS and IAPT.
   b. Increased assessment of both maternal and paternal mental health, for example by using questionnaire measures.
   c. Psycho-education for parents around their own mental health and where to seek help as part of treatment package.
   d. Increased recording of whether parents are receiving treatment.
   e. Identifying ways of improving communication and joined-up working between adult and child mental health services.
3. Increased use of questionnaires for assessment
   a. Ensure questionnaires are readily available to staff.
4. Always offer psychological intervention (CBT, IPT or brief FT) as first line treatment.
   a. Investigate and address the reasons why no cases were receiving IPT alone.
5. When first prescribing medication, see client weekly for at least first four weeks.
6. Language and use of interpreters:
   a. Highlight need to record first language
   b. Remove default setting that interpreter is not needed.

7. Dissemination

The audit will be presented to the National and Specialist Mood Disorders team at the Michael Rutter Centre, the CAMHS Executive which has attendance from all boroughs and N&S teams and to the Audit committee. See Appendix 2 for a brief summary of the audit findings which will be disseminated.

8. References

Ahmed, I. (2001). Psychological aspects of giving and receiving medications. In Tseng, W.-S., & Streltzer, J. (Eds.). *Culture and psychopathology: A guide to clinical practice.* Washington, DC: American Psychiatric Association.

Angold, A., & Costello, E.J. (1995) A test-retest reliability study of child-reported psychiatric symptoms and diagnoses using the Child and Adolescent Psychiatric Assessment (CAPA-C). *Psychological Medicine,* 25, 755–762.

Angold, A., & Costello, E.J. (2001). The epidemiology of depression in children and adolescents. In I. M. Goodyer (ed.) *The Depressed Child and Adolescent*, 2nd edn. Cambridge Child and Adolescent Psychiatry series. Cambridge: Cambridge University Press.

Angold, A., Costello, E.J., Messer, S.C., Pickles, A., Winder, F., & Silver, D. (1995). The development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*, 5, 237–249.

Angold, A., Erkanli, A., Silberg, J., Eaves, L., & Costello, J. (2002) Depression scale scores in 8–17 year-olds: effects of age and gender. *Journal of Child Psychology and Psychiatry,* 43, 1052–1063.

Angold, A., & Egger, H.L. (2004) Psychiatric diagnosis in preschool children. In: DelCarmen-Wiggins, R. & Carter, A. (eds.) *Handbook of Infant, Toddler, and Preschool Mental Health Assessment* (pp. 123–139). New York: Oxford University Press.

Brent, D., Kolko, D., Birmaher, B., Baugher, M., Bridge, J., Roth, C.. & Holder, D. (1998) Predictors of Treatment Efficacy in a Clinical Trial of Three Psychosocial Treatments for Adolescent Depression. *Journal of the American Academy of Child & Adolescent Psychiatry,* 37, 906-914.

Brent, D., Perper, J., Moritz, G., Allman, C., Friend, A., Roth, C., Schweers, J., Balach, L., & Baugher, M. (1993) Psychiatric risk factors for adolescent suicide: a case-control study. *Journal of the American Academy of Child and Adolescent Psychiatry,* 32,11–16.

Carr, A. (2006) *Handbook of child and adolescent clinical psychology 2nd ed*. London: Routledge.

Carr, A. (2008) Depression in young people: Description, assessment and evidence-based treatment. *Developmental Neurorehabilitation,* 11, 3–15

Cochran, S. V., & Rabinowitz, F. E. (2000) *Men and Depression: Clinical and Empirical Approaches.* San Diego: Academic Press.

Cohen, P., Kasen, S., Brook, J.S., & Struening, E. (1991) Diagnostic predictors of treatment patterns in a cohort of adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 989–993

Committee on Safety of Medicines (CSM) (2003). Selective serotonin reuptake inhibitors (SSRIs): overview of regulatory status and CSM advice relating to major depressive disorder (MDD) in children and adolescents including a summary of available safety and efficacy data.

Coyle, J.T., Pine, D.S., Charney, D.S., Lewis, L., Nemeroff, C.B., Carlson, G. A., Toor Joshi, P., Reiss, D., Todd, R.D., & Hellander, M. (2003) Depression and bipolar support alliance consensus statement on the unmet needs in diagnosis and treatment of mood disorders in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 1494–1503.

Dent, T.H.S., & Sadler, M. (2002) From guidance to practice: Why NICE is not enough. *British Medical Journal, 324,* 842

Emslie, G.J., Mayes, T.L., Laptook, R.S., & Batt, M. (2003) Predictors of response to treatment in children and adolescents with mood disorders. *Psychiatric Clinics of North America, 26,* 435–456.

Fombonne, E., Wostear, G., Cooper, V., Harrington, R., & Rutter, M. (2001a) The Maudsley long-term follow-up of child and adolescent depression: 1. Psychiatric outcomes in adulthood. *British Journal of Psychiatry,* 179, 210–217.

Fombonne, E., Wostear, G., Cooper, V., Harrington, R., & Rutter, M. (2001b) The Maudsley long-term follow-up of child and adolescent depression: 2. Suicidality, criminality and social dysfunction in adulthood. *British Journal of Psychiatry, 179,* 218–223.

Goldberg, D. P. (1972). *The Detection of Psychiatric Illness by Questionnaire. Maudsley Monograph No. 21*. Oxford University Press: London.

Goodyer, I.M., & Cooper, P.J. (1993) A community study of depression in adolescent girls. II: The clinical features of identified disorder. *British Journal of Psychiatry, 163,* 374–380.

Green, H., McGinnity, A., Meltzer, H., Ford, T., & Goodman, R. (2004) *Mental health of children and young people in Great Britain.* Palgrave Macmillan, London

Grimshaw, J.M., Eccles, M.P., Walker, A.E., & Thomas, R. (2002) Changing physician's behaviour: what works and thoughts on getting more things to work. *The Journal of Continuing Education in the Health Professions, 22,* 237–243.

Hazell, P., O'Connell, D., Heathcote, D., & Henry, D. (2002). Tricyclic drugs for depression in children and adolescents. *Cochrane Database of Systematic Reviews 2002, Issue 2.*

Healy, D. (2003) Lines of evidence on the risks of suicide with selective serotonin reuptake inhibitors. *Psychotherapy and Psychosomatics,* 72, 71–79.

Herbert, J., Goodyer, I.M., Altham, P.M., Pearson, J., Secher, S.M., & Shiers, H.M. (1996) Adrenal secretion and major depression in 8- to 16-year-olds. II. Influence of co-morbidity at presentation. *Psychological Medicine, 26,* 257–263.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., Williamson, D., & Ryan, N. (1997) Schedule for Affective Disorders and Schizophrenia for School-Age Children-

Present and Lifetime Version (K-SADS-PL): initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 980–988.

Keenan, K., Shaw, D.S., Walsh, B., Delliquadri, E., & Giovannelli, J. (1997) DSM-III-R disorders in preschool children from low-income families. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 620–627.

Kolvin, I., & Sadowski, H. (2001). Childhood depression: clinical phenomenology and classification. In Goodyer, I. M. (ed.) *The Depressed Child and Adolescent* (pp. 233–266). Cambridge Child and Adolescent Psychiatry series. Cambridge: Cambridge University Press.

Kovacs, M., Gatsonis, C., Paulauskas, S., & Richards, C. (1989).Depressive disorders in childhood. IV. A longitudinal study of comorbidity and risk for anxiety disorders. *Archives of General Psychiatry,* 46, 776-782.

Kroenke, K., Spitzer, R.L., & Williams, J.B.W (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, 16, 606-613.

Lau, A. S., Jernewall, N. M., Zane, N., & Myers, H. F. (2002) Correlates of suicidal behaviours among Asian-American outpatient youths. *Cultural Diversity and Ethnic Minority Psychology, 8,* 199–213.

Lavigne, J.V., Arend, R., Rosenbaum, D., Binns, H.J., Christoffel, K.K., Burns, A., & Smith, A. (1998) Mental health service use among young children receiving paediatric primary care. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 1175–1183.

Lavigne, J.V., Gibbons, R.D., Christoffel, K.K., Arend, R., Rosenbaum, D., Binns, H., Dawson, N., Sobel, H., & Isaacs, C. (1996) Prevalence rates and correlates of psychiatric disorders among preschool children. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 204–214.

Luby, J.L., Heffelfinger, A.K., Mrakotsky, C., Brown, K., Hessler, M., Wallis, J.M., & Spitznagel, E.L. (2003). The clinical picture of depression in preschool children. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 340–348.

Luby, J., Heffelfinger, A., Mrakotsky, C., Hessler, M., Brown, K., & Hildebrand, T. (2002). Preschool major depressive disorder: Preliminary validation for developmentally modified DSM-IV Criteria*. Journal of the American Academy of Child and Adolescent Psychiatry, 41,* 928–937.

Malek, M., & Joughin, C. (eds) (2004) *Mental Health Services for Minority Ethnic Children and Adolescents*. London: Jessica Kingsley.

Meltzer, H., Gatward, R., Goodman, R., & Ford, T. (2000). *The Mental Health of Children and Adolescents in Great Britain*. London: The Stationery Office

Mitchell, J., McCauley, E., Burke, P.M., & Moss, S.J. (1988) Phenomenology of depression in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 27,* 12–20.

National Institute for Clinical excellence (2005) *CG28 Depression in Children and Young People: Full guidance.* Available at http://guidance.nice.org.uk/CG28/Guidance

Post, R.M. (1992) Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. *American Journal of Psychiatry, 149,* 999–1010.

Rawlins, M.D. (2004) NICE work – Providing Guidance to the British National Health Care Service. *The New England Journal of Medicine, 351, 14*

Real, T. (1997) *I Don't Want to Talk About it: Overcoming the Secret Legacy of Male Depression.* New York: Simon & Schuster.

Rhodes, L.A., Genders, R., Owen, R., O'Hanlon, K., & Brown, J.S.L. (2010) Investigating barriers to implementation of the NICE Guidelines for Depression: a staff survey with Community Mental Health Teams *Journal of Psychiatric and Mental Health Nursing, 17,* 147–151

Ryan, N.D., Puig-Antich, J., Ambrosini, P., Rabinovich, H., Robinson, D., Nelson, B., & Iyengar, S. (1987) The clinical picture of major depression in children and adolescents. *Archives of General Psychiatry,* 44, 854–861.

Saxe, L., Cross, T., & Silverman, N. (1988) Children's mental health: the gap between what we know and what we do. *American Psychologist,* 43, 800–807

Sokolov, S., & Kutcher, S. (2001) Adolescent depression: Neuroendocrine aspects. In Goodyer, I. M. (ed.), *The Depressed Child and Adolescent (*pp. 233–266). Cambridge Child and Adolescent Psychiatry series. Cambridge: Cambridge University Press.

Spitzer, R.L., Kroenke, K., Williams, J.B.W., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder: the GAD-7. *Archives of Internal Medicine,* 166, 1092-1097.

Thomas, C.M., & Morris, S. (2003) Cost of depression among adults in England in 2000. *British Journal of Psychiatry,* 183, 514–519.

Wu, P., Hoven, C.W., Bird, H.R. Moore, R.E., Cohen, P., Alegria, M., Dulcan, M.K., Goodman, S.H., Horwitz, S.M., Lichtman, J.H., Narrow, W.E., Rae, D.S., Regier, D.A., & Roper, M.T. (1999) Depressive and disruptive disorders and mental health service utilization in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry,* 38, 1081–1090

Wu, P., Hoven, C.W., Cohen, P., Liu, X., Moore, R.E., Tiet, Q., Okezie, N., Wicks, J., & Bird, H. (2001) Factors Associated with Use of Mental Health Services for Depression by Children and Adolescents. *Psychiatric Services,* 52, 189-195

9. Appendix

Appendix 1: Additional Results

Table A: Expected number of cases per borough compared to the number of cases seen in borough.

	Age group	Expected number of cases	Number of cases in borough	Percentage of cases seen
CROYDON	0 to 11	549	7	1.28%
	12 to 18	921	231	25.08%
LAMBETH	0 to 11	405	17	4.20%
	12 to 18	493	165	33.47%
LEWISHAM	0 to 11	430	1	0.23%
	12 to 18	583	160	27.44%
SOUTHWARK	0 to 11	407	14	3.44%
	12 to 18	522	175	33.52%

Table B: Population data by gender

ONS	Male	Female	Ratio (Female/Male)
0 to 18	128989	134111	1.04
0 to 11	91271	87865	0.96
12 to 18	42840	41124	0.96

Table C: Sample data by gender

Sample	Male	Female	Ratio (Female/ Male)
0 to 18	257	513	2.00
0 to 11	26	13	0.5
12 to 18	231	500	2.16

Table D: Sex ratios within each borough

	Age group	Male	Female	Ratio (female/male)
CROYDON	0 to 11	27,965	26,957	1.04
	12 to 18	15,876	14,828	1.07
LAMBETH	0 to 11	20,547	19,918	1.03
	12 to 18	8264	8,180	1.01
LEWISHAM	0 to 11	22,048	20,978	1.05
	12 to 18	9787	9,634	1.02
SOUTHWARK	0 to 11	20,711	20,012	1.03
	12 to 18	8913	8,482	1.05

Tables E &F: Sex ratio in each borough for each age group for sample.

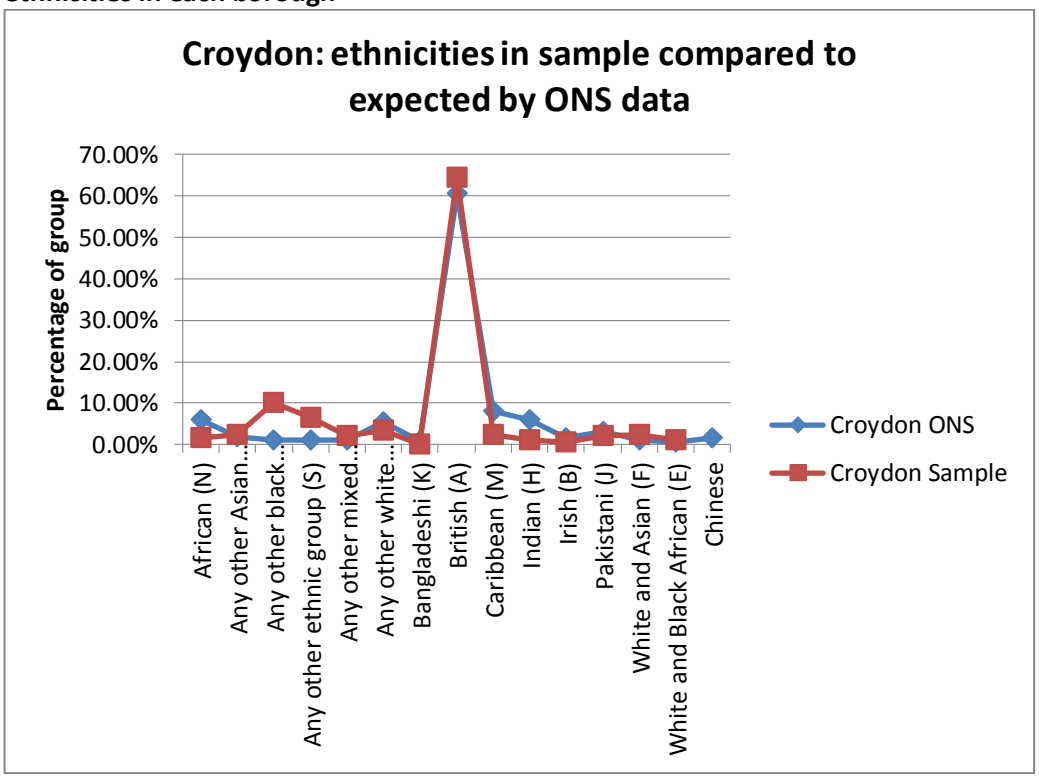
0 to 11	male	female	ratio
Croydon	4	3	0.75
Lambeth	13	4	0.31
Lewisham	1	0	0
Southwark	8	6	0.75

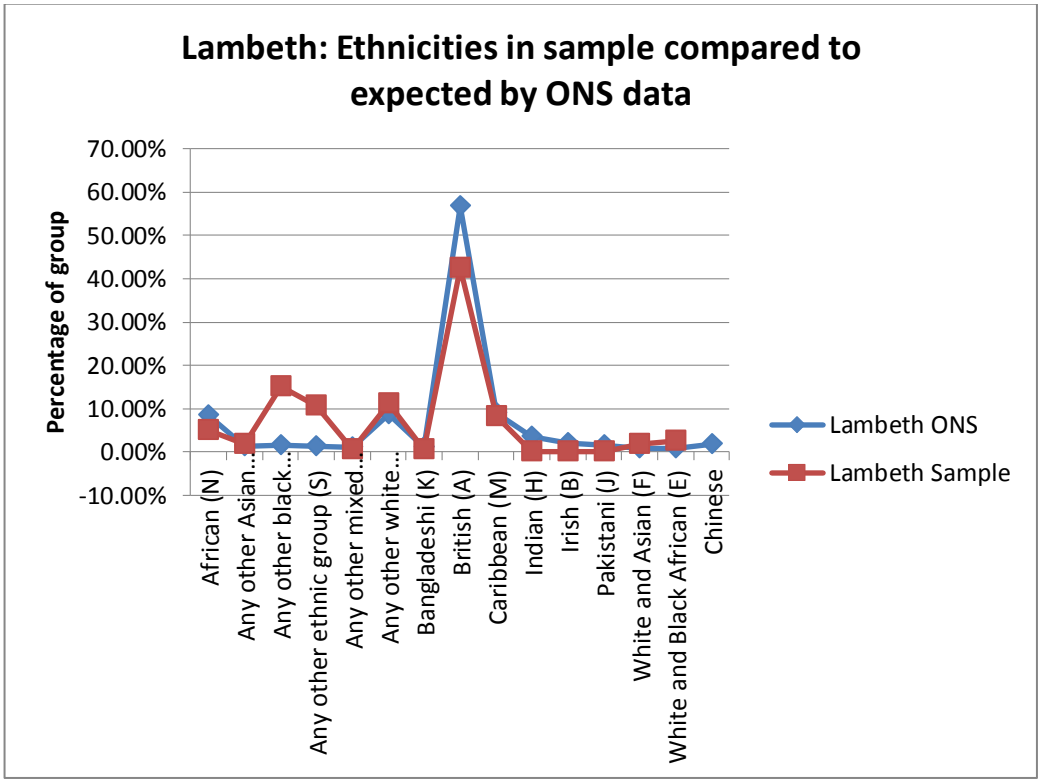
12 to 18	Male	Female	ratio
Croydon	81	150	1.85
Lambeth	64	101	1.58
Lewisham	44	116	2.64
Southwark	42	133	3.17

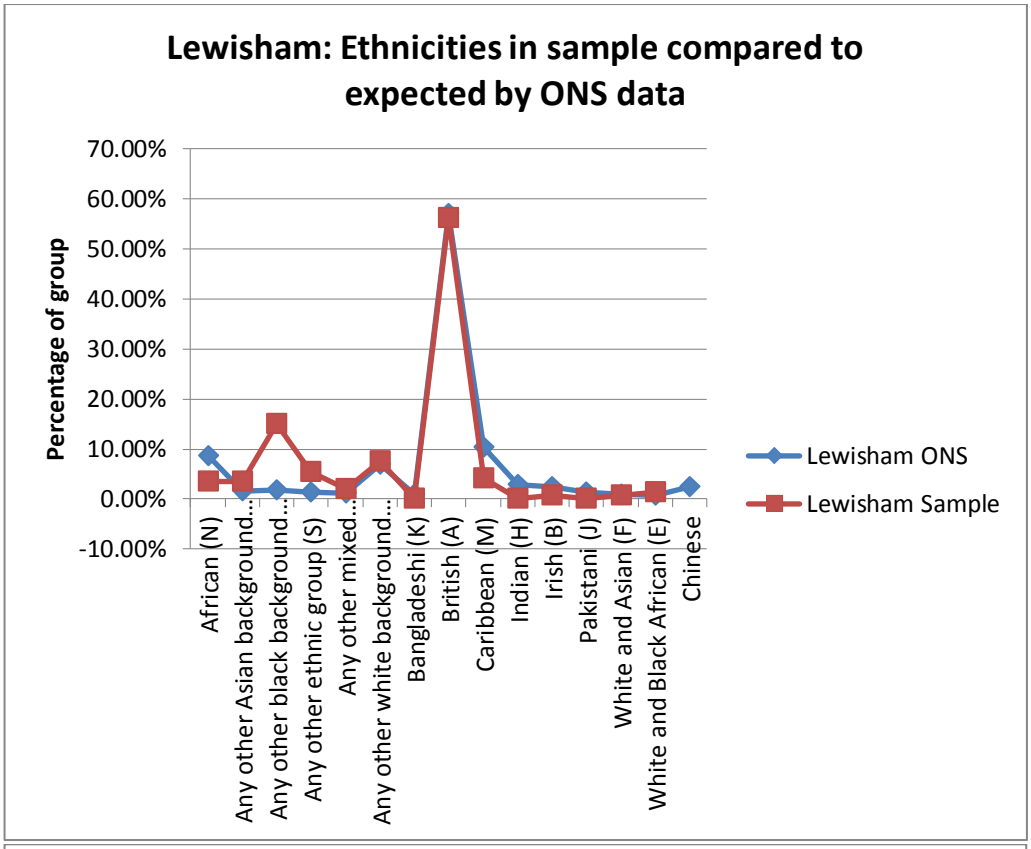
Table G: Ethnicities in across boroughs compared to group data

		Sample	
	% ethnicity in SLAM boroughs (ONS)	**Percentage of sample**	**% not including missing and not stated**
African (N)	8.18%	4.68%	5.30%
Any other Asian background (L)	1.47%	2.21%	2.50%
Any other black background (P)	1.44%	11.69%	13.25%
Any other ethnic group (S)	1.39%	6.75%	7.66%
Any other mixed background (G)	1.05%	1.17%	1.33%
Any other white background (C)	7.12%	5.84%	6.63%
Bangladeshi (K)	1.10%	0.26%	0.29%
British (A)	57.70%	47.01%	53.31%
Caribbean (M)	8.49%	4.55%	5.15%
Indian (H)	4.21%	0.26%	0.29%
Irish (B)	2.03%	0.39%	0.44%
Pakistani (J)	2.04%	0.52%	0.59%
White and Asian (F)	0.98%	1.30%	1.47%
White and Black African (E)	0.66%	1.56%	1.77%
Chinese	2.13%	0%	0%
missing ethnicity	NA	10.00%	NA
Not Stated (Z)	NA	1.82%	NA

Figures A to D: graphs illustrating the ethnicities within the group compared to the expected ethnicities in each borough






Lewisham: Ethnicities in sample compared to expected by ONS data
Percentage of group
70.00%
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
-10.00%
African (N)
Any other Asian background...
Any other black background...
Any other ethnic group (S)
Any other mixed...
Any other white background...
Bangladeshi (K)
British (A)
Caribbean (M)
Indian (H)
Irish (B)
Pakistani (J)
White and Asian (F)
White and Black African (E)
Chinese
Lewisham ONS
Lewisham Sample

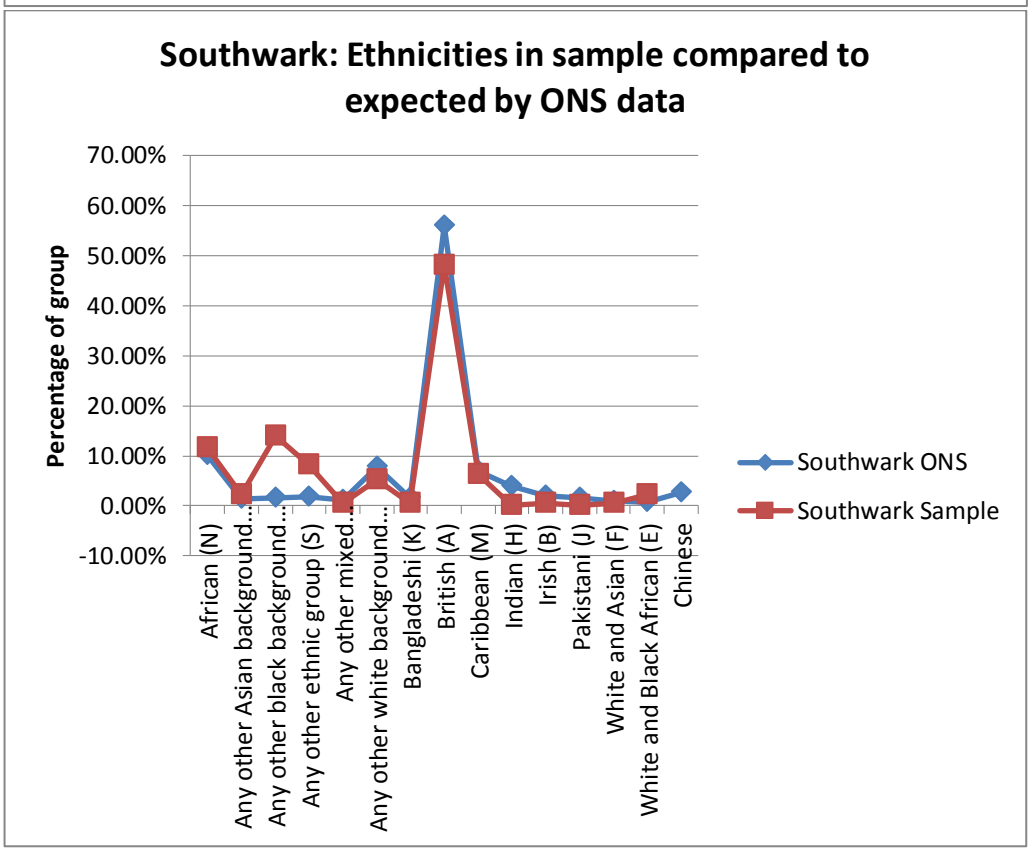

Southwark: Ethnicities in sample compared to expected by ONS data
Percentage of group
70.00%
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
-10.00%
African (N)
Any other Asian background...
Any other black background...
Any other ethnic group (S)
Any other mixed...
Any other white background...
Bangladeshi (K)
British (A)
Caribbean (M)
Indian (H)
Irish (B)
Pakistani (J)
White and Asian (F)
White and Black African (E)
Chinese
Southwark ONS
Southwark Sample

Figure E: Number accepting psychological intervention

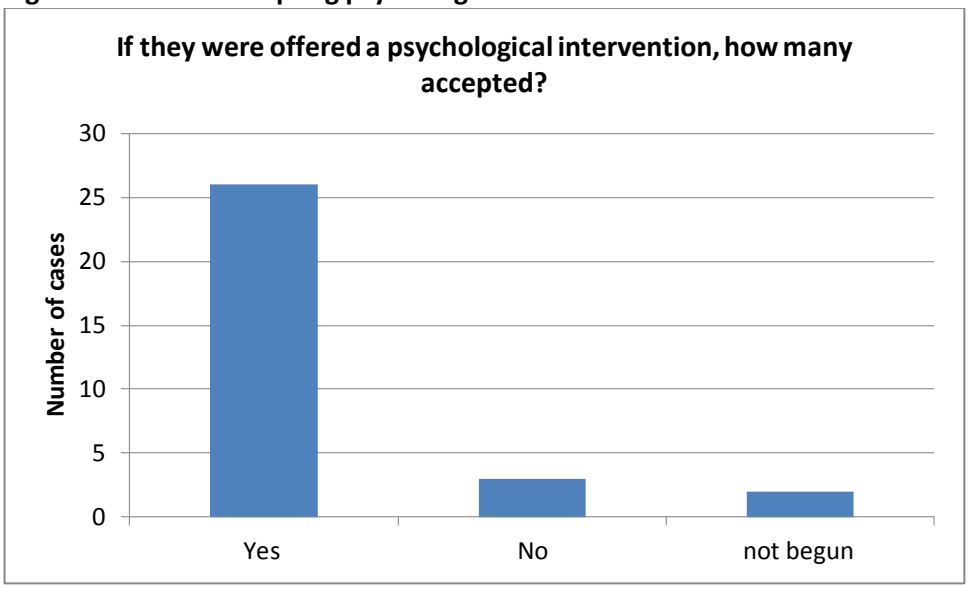


Figure F: Number of psychological interventions offered

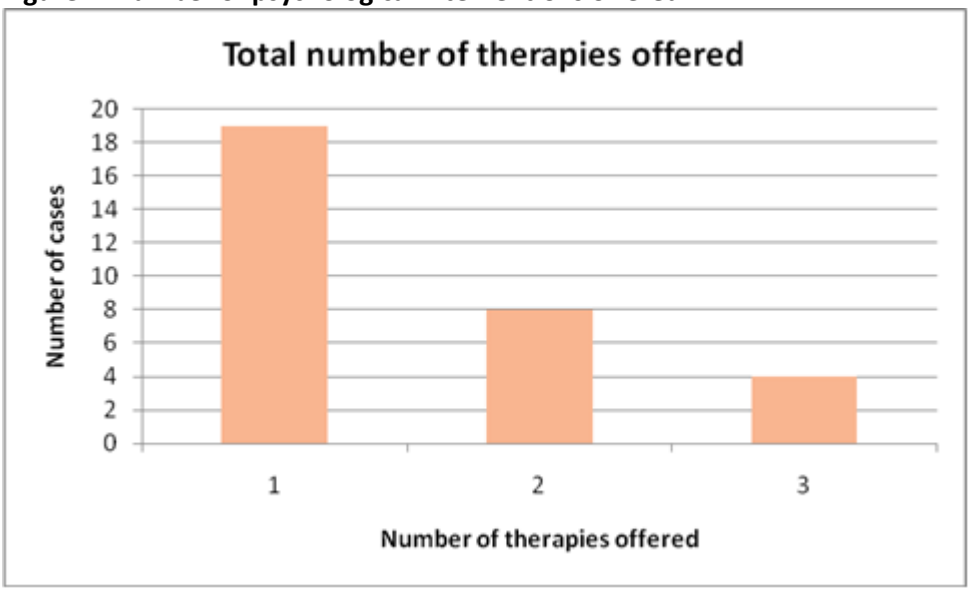
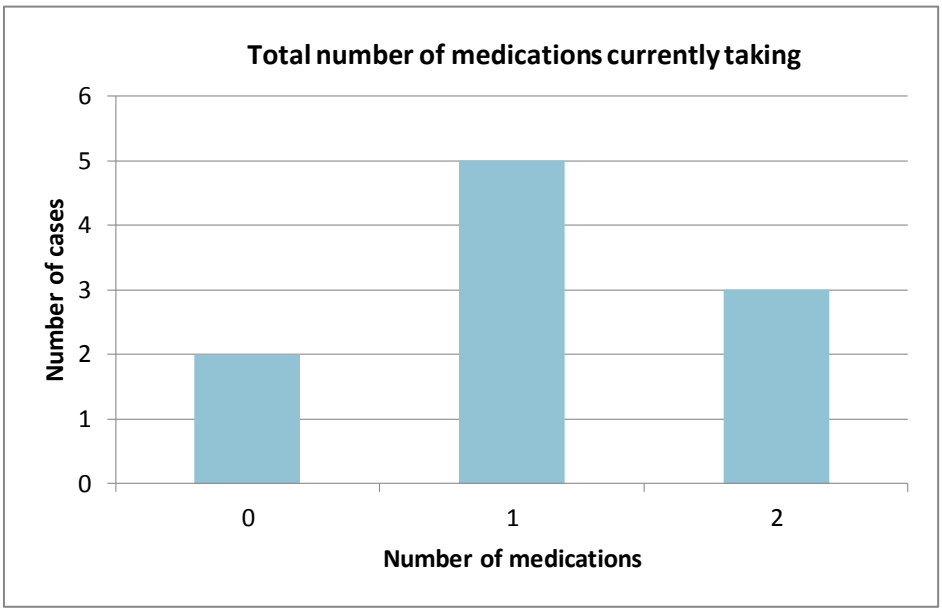


Figure G: Number of medications cases are currently taking



Appendix 2: Brief summary of findings for dissemination

Brief Summary of findings

Supervised by Dr Patrick Smith
2012

Depression in Children and Young People: How Compliant is SLAM with NICE Guidelines?

Aims
This audit consists of two main questions:
Part 1: Who is presenting to SLAM?

- Gather demographic and service information on the C&YP presenting to SLAM with depression in a one-year period.
- Compare the cases seen by SLAM to what would be expected according to population figures and prevalence rates.

Part 2: Is SLAM compliant with NICE guidelines?

- Evaluate the level of adherence in SLAM to NICE guidelines when treating C&YP with depression.
- Validate good adherence and highlight areas where adherence or recording of information is lacking.

Methodology: the Case Register Interactive Service (CRIS) was used to gather anonymised records of all C&YP presenting to SLAM with depression over a one-year period.
Part 1: the number and demographic spread of these cases was compared to what would be expected from prevalence and population data.
Part 2: the sample was stratified according to age, sex, ethnicity and borough and a random sample of 5% of cases (45 cases) was taken that reflected the spread of data identified by the stratification. These cases were then analysed and rated against NICE guidelines.

Conclusions:
Part 1: The results indicate that there is a large discrepancy between the expected number of cases of depression in the population and the number of cases seen by SLAM, especially in the 0 to 11 age group.

1. Number of cases: SLAM is seeing 2% of estimated cases of depression in the 0 to 11 age group and 29% of those in the 12 to 18 age group. No children aged 2 to 5 with depression were being seen in SLAM at the time of data collection
2. Sex ratio (female: male): a ratio of 1:2 is seen in the under 11s (expected ratio 1:1) and just over 2:1 in the 12 to 18 age group (expected ratio 2:1).
3. Ethnicity: 'Any other black background' and 'any other ethnic group' may be over-represented in the sample whilst 'Indian' may be under-represented in the sample.

Part 2: In general, adherence to NICE guidelines is good but there are areas that require improvement. Adherence to NICE guidance in terms of risk assessment and recording of comorbidities was good. Areas where compliance was lacking included the recording of first language, parental mental health assessment and treatment, use of a depression specific questionnaire, use of counselling/supportive therapy as a first line intervention and monitoring of medication.

Recommendations

1. Dissemination of audit findings through written reports, presentations at meetings and discussion with professionals.
2. Consideration of ways to make guidelines more accessible and relevant to CAMHS professionals, for example having a brief summary of the key guidelines and having meetings with CAMHS staff to discuss the guidelines and why they are important.

Part 1: Who is presenting to SLAM?

1. Increased recognition and assessment of depression across age groups especially in the under 11 age group. Consideration is required concerning which combination of teams would deliver the following:
    a. Psycho-education on symptoms and treatment of depression in schools and tier 1.
    b. Increasing parental awareness of emotional difficulties in C&YP to enhance early identification and treatment, for example leaflets targeted at parents and placed in GP surgeries and schools.
2. Increased understanding by professionals of the different presentation of depression in males and females, for example through time allocated to Continued Professional Development.
3. Further investigation of referral patterns for Black and Minority Ethnic groups, for example through future audit.

Part 2: Is SLAM compliant with NICE guidelines?

1. Epjs
    a. Clear guidance on mandatory fields in ePJS and rationale for these fields e.g. impact of parental mental health on outcomes.
    b. Consider functionality within ePJS to remind clinicians when key fields are not completed, for example automated reminders or highlighted boxes.
    c. NICE guidance integrated more fully into ePJS e.g. flow chart for NICE recommendations when depression diagnosis is entered.
2. Parental mental health
    a. More integrated treatment of adult and child mental health issues. For example, direct referral pathways and heightened communication between CAMHS and IAPT.
    b. Increased assessment of both maternal and paternal mental health, for example by using questionnaire measures.
    c. Psycho-education for parents around their own mental health and where to seek help as part of treatment package.
    d. Increased recording of whether parents are receiving treatment.
    e. Identifying ways of improving communication and joined up working between adult and child mental health services.
3. Increased use of questionnaires for assessment
    a. Ensure questionnaires are readily available to staff.
4. Always offer psychological intervention (CBT, IPT or brief FT) as first line treatment.
    a. Investigate and address the reasons why no cases were receiving IPT alone.
5. When first prescribing medication, see client weekly for at least first four weeks.
6. Language and use of interpreters:
    a. Highlight need to record first language
    b. Remove default setting that interpreter is not needed.